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SUPPLEMENTUM 2

# Electrical Burns

*A Clinical and Experimental Study*
*with Special Reference*
*to Peripheral Nerve Injury*

By OLE M. UGLAND

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PLASTIC AND RECONSTRUCTIVE SURGERY
SUPPLEMENTUM 2

*From the Department of Plastic Surgery*
*and the Laboratory of Clinical Neurophysiology,*
*University Hospital, Uppsala, Sweden*

# Electrical Burns

## *A Clinical and Experimental Study with Special Reference to Peripheral Nerve Injury*

By OLE M. UGLAND

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# I. CLINICAL STUDY

## INTRODUCTION

The first victim of industrial electricity is believed to have been a carpenter in Lyon who was killed in 1879 by alternating current at 250 V from a Siemens dynamo (Jex-Blake, 1913), and up to 1891 only 10 deaths from electricity were known (Naville & de Morsier, 1927).

During the following years with the steadily increasing production of electrical energy there were a rapidly increasing number of deaths from electricity. In England 183 such deaths occured during the period 1901–1910 (Jex-Blake, 1913) and in France alone 571 accidents with a mortality rate of 4.9% were reported in 1907 (Naville & de Morsier, 1927).

As early as 1921 Jaeger expressed the view that the number of accidents in Switzerland did not keep pace with the production of electrical energy, and a similar tendency in Germany during the late twenties was reported by Alvensleben (1939). Although the figures still ran high there apparently was a relative decrease which may be attributed to broader experience and improvement in safety precautions.

Reliable statistics on electrical injuries are difficult to obtain as many accidents are not reported to the Board of Health in the different countries. Furthermore, burns from electricity are sometimes, in official statistics, classified as burns only, without regard to the causative agent. Hughes (1956) reported from England an annual number of 750 accidents, the mortality rate being 7.5%. He claimed that the increased use of electricity, in industry at least, had resulted in a relative fall in accident and mortality rates; at the turn of the century 17% of industrial electrical accidents were fatal compared with 6% between 1920 and 1930, and 4% in 1956. At present about 300 accidents occur each year in Switzerland and the mortality rate is 10% (Lang & Baur, 1960). According to Poticha *& al.* (1962) and Snyder (1963) the number of accidents in the United States is increasing, but they give no figures. Other authors state that approximately 1000 persons are killed by electricity each year in the United States (Lewis, 1958; Silversides, 1964; Robinson *& al,* 1965; Mills *& al.* 1966). In Denmark, 666 accidents, 92 of which were fatal, were reported during the period 1954–1963 (Machholdt, 1965). In Norway the production of electrical energy has increased more than 20 times during the last 50 years, but despite this the annual number of deaths from electricity has remained constant. (Annual average 8.6 persons.)

Sweden is ranked as no. 4 (close to the United States) in the production of electrical energy, counted per inhabitant. Figure 1 shows the annual incidence of accidents plotted against the production of electricity. Two characteristics of this graph should be stressed: One is the fact that the mortality rate has remained approximately the same during the last 50 years. Admittedly, the numbers are small, but it seems difficult to reduce the mortality rate in spite of recent advances in the treatment of electrical injuries. Another point is the marked decrease in the accident rate during the last 10 years although the production of electrical energy has been increased.

Although electrical burns are generally classified as thermal burns, they present special problems. Burns from electricity more often have a prolonged healing time and result in more crippling deformities than do other thermal burns. The literature on this subject consists of solitary case reports or general surveys but the reports on clinical material are, with several exceptions

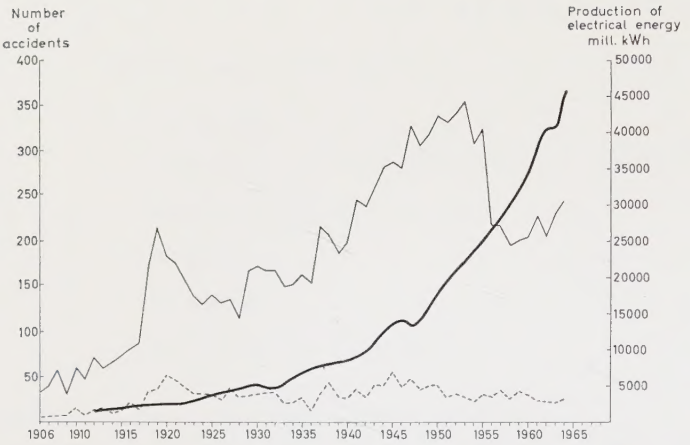



*Fig. 1.* Number of injuries and deaths from electricity plotted against the production of electrical energy in Sweden during the last 60 years.

———, Number of accidents;
– – – –, number of deaths;
———, production of electrical energy,

brief, comprising small numbers of patients. These reports will be referred to at relevant places in the following text.

During the period 1951 to 1967, 122 patients with electrical burns have been treated in the Burns Unit, Department of Plastic Surgery at the University Hospital of Uppsala, Sweden. This material is unique in many respects; a detailed clinical study of such a large number of cases has not been reported before. Information concerning the type of current, voltage, duration of contact and the pathway of the current are recorded in these cases, thus permitting a reliable reconstruction of the accident. The principles of treatment have been fairly unchanged during the period of investigation, and the patients have been subject to careful follow up studies, most of them over many years.

The purpose of this investigation is to report on this material, to give a thorough analysis of the local and general lesions in relation to technical data available in these cases, to report on principles of treatment, incapacity for work and permanent disability. Before presenting the clinical material in detail, basic principles in physics which affect the lesions produced by electricity will be discussed.

## TECHNICAL ASPECTS OF ELECTRICAL INJURIES

The damage inflicted upon the human body by electricity depends on various factors such as amperage, voltage, resistance, the type of current and its pathway through the body, duration of current flow and the surface area of contact.

### *1. Amperage*

While hazards from electricity are partly dependant upon the voltage, the actual danger to life depends mainly on the amperage. It is the current flow that gives rise to tissue damage. Since the amperage is a function of the voltage and the resistance it would seem easy to estimate its value in a person exposed to electricity. This is, however, not possible as we do not know his resistance at the moment of the accident. Such calculations are therefore worthless.

Animal experiments indicate that—as far as alternating current at 50 cycles/second is concerned—a current intensity at 100 mA for 1–2 seconds duration means certain death, provided the heart is traversed by the current (Jex-Blake, 1913; Löbl, 1933; Kouwenhoven, 1949; Koeppen, 1955; Schliephake & Smets, 1963 and others). Most authors agree that the lethal amount of direct current is three to four times that of alternating current.

### *2. Voltage*

In most countries voltages of less than 500 V are classified as low tension. In Sweden the borderline between low- and high tension is set at 380 V. A lethal voltage cannot be exactly fixed. Although shocks of 50,000 V or more may be survived, a low tension shock may cause instant death. According to Jellinek (1934) voltages over 40 V should be considered dangerous.

For reasons of safety, electrical energy is distributed to the household at relatively low voltages and the present tendency is to standardize on 220 V alternating current for lighting and domestic service.

In electrical accidents we usually know the voltage, but it should be kept in mind when dealing with three-wire systems (Fig. 2) that the rated voltage is not necessarily the actual voltage to which the victim has been exposed. Thus the voltage between each outside wire and the neutral is: Rated voltage $/\sqrt{3}$.

Strictly speaking, the formula $V = R \cdot I$ is not applicable to the human body (Jellinek, 1902, 1934). This is mainly because the skin acts as a capacitive resistance (Gildemeister, 1919; Freiberger, 1934; Osypka, 1963). As ex-

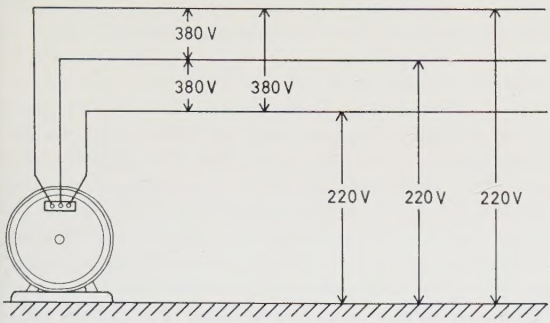



*Fig. 2.* Potentials between different phases and earth in a 380 V three wire system.

perienced electropathologists like Koeppen (1955) and others have not doubted the validity of Ohm's law in the human body, it seems justifiable to use the formula for making the following generalization: The higher the voltage, the higher the amperage, provided the resistance of the victim is relatively constant. Clinical experience confirms that the risk of severe burns is proportional to the voltage.

The extensive burns often seen after high tension accidents are partly caused by arcing of the current. The arc has an intense pale violet light and consists of ionized air particles, which maintain electrical continuity. Much of the electrical energy is consumed in the arc and the voltage drop is considerable (Mueller, 1940; Jenny, 1945). The extremely high temperature in an arc (up to 4000°C) can melt bone and volatilize metal. Under ordinary atmospheric conditions, arcing will amount to several centimeters for each 10,000 V. These values should be regarded as approximate because accounts of spark distance are somewhat contradictory (Prevost & Batelli, 1899; Mueller, 1940; Brown & Moritz, 1964). Once a high voltage arc is established, it can be extended to about ten feet before interupting the circuit (Brown & Moritz, 1964).

Considering the immediate results of an electrical injury upon vital functions of the body, the significance of the voltage is not yet completely understood. In low tension accidents the cause of death is supposed to be cardiac arrest or ventricular fibrillation (Prevost & Batelli, 1899; Jex-Blake, 1913; Fischer & Fröhlicher, 1951; Kouwenhoven, 1949). High tension is believed to kill through destruction or inhibition of the respiratory centre (Prevost & Batelli 1899; Maclachlan, 1930; Pearl, 1933; Kouwenhoven, 1949), but as the heart sometimes continues to beat, resuscitation may prove successful. Koeppen & *al.* (1962) however, maintain that immediate death by electrical shock is always a cardiac death.

### *3. Resistance*

Calculations of resistance and current flow in electrical injuries are of minor practical importance, but they are of great theoretical interest.

A person completing a current circuit between two contact points has a total resistance composed of several resistors: The skin resistance at one contact point + the internal body resistance + the skin resistance at the other contact point. In addition there is often the resistance of foot wear, gloves, clothing etc. at one or both contact points.

*(a) Skin resistance.* The skin resistance varies within wide limits; not only from one person to another, but also at different places of the same person, depending upon thickness, moisture and cleanness of the skin. Freiberger (1934) showed that the skin resistance is particularly related to the stratum corneum of the epidermis which acts as an insulator to the internal body, and he compared the skin to the dielectric in a condenser.

The voltage gradient in an insulator cannot be increased indefinitely. There is a critical value at which the material ceases to act as an insulator and "breaks down". From a technical point of view the skin is a poor insulator and its break down occurs at comparatively low voltages. Thus Freiberger produced break down in cadaver skin at 700 V. However, the skin resistance increases rapidly after death and he maintains that the practical significance of the skin as an insulator is restricted to the range under 250 V. The skin offers greater resistance to direct than to alternating current. (Bachem, 1930; Freiberger, 1934), and the resistance is inversely proportional to the frequency (Osypka, 1963).

From a practical point of view it is important to know that the resistance of a dry palm may be as high as 2 megohm (Jellinek, 1902), and if wet, the resistance may be reduced a thousandfold.

*(b) Internal body resistance.* Following a complete break down of the skin at the contact points we may take only the internal body resistance into consideration. This is a pure Ohmic resistance which—at least within the range of 5 to 5000 V—is relatively constant between 500 and 1000 ohm, depending on the location of the contacts (Freiberger, 1934).

Freiberger and Osypka (1963) state that the internal body resistance is manly confined to the limbs, because of their contents of high resistant bones, joint and tendons. Jellinek (1902) gave the resistivity of different tissues in the following decreasing order: Bone, cartilage, nerve, tendon, thyroid gland, lung, spleen, epicardium, myocardium, brain, muscle, kidney, liver. The body fluids are supposed to have approximately the same resistivity as kidney. These figures differ somwhat from the statement by Dale (1954), Robinson & *al.* (1965) and others, but all agree that bone and tendons have the highest resistivity. If we assume that the amount of the internal body resistance is largely determined by the limbs, the resistance of the trunk becomes negligible in comparison. For the sake of simplicity we may then regard the human body as a network of equal large resistors in series and in parallel (Osypka, 1963). A rough estimation of the internal body resistance between different contact points is shown in figure 3, A and B. If we call the resistance 100% between both hands it should be reduced to 50% between both hands and both feet. Consequently the amperage will be higher, thus increasing the danger of disturbances of the heart function.

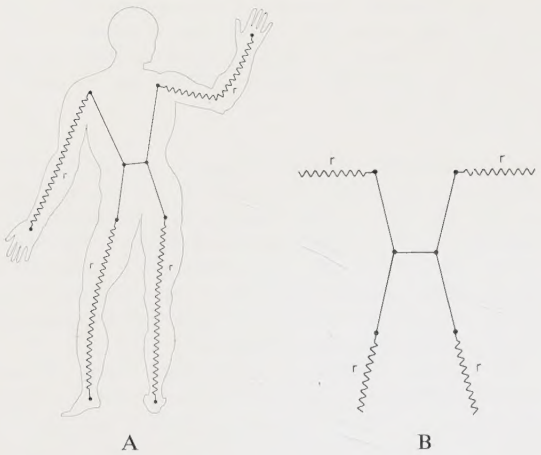



*Fig. 3 A, B.* Circuit diagrams of internal body resistance along different current pathways.

Hand—body—hand: $R = r + r = 2r = (100\%)$

Hand—body—foot: $R = r + r = 2r = (100\%)$

Hand—body—feet: $R = r + r_1 = 1.5r = (75\%)$

$$\frac{1}{r_1} = \frac{1}{r} + \frac{1}{r}$$

$$r_1 = 0.5r$$

Hands—body—feet: $R = r_1 + r_2 = 1r = (50\%)$

$$\frac{1}{r_2} = \frac{1}{r} + \frac{1}{r}$$

$$r_2 = 0.5r$$

*4. Type of current*

Alternating current has now superseded direct current because it is cheap and can easily be transformed into any voltage. The frequency is commonly expressed in cycles/second. Alternating current in the ordinary distribution network in Sweden has a frequency of 50 c/s, while the railway systems use $16^{2}/_{3}$ c/s. The use of direct current is mainly limited to chemical and metallurgical industries and to street-car systems; the vast majority of electrical injuries are caused by alternating current.

That alternating current is three to four times as dangerous as direct current is the prevailing opinion. This is however not always true. Killinger (1961) called attention to the high mortality rate in electrical accidents in German ore mines, where the transport trains are driven by direct current at 220–250 V. The distance between the adit floor and the live wire running under the ceiling is comparatively short and the wire may easily be touched by a man's head. The current thus can pass to the ground through the long axis of the body. Although the voltage does not seem dangerous, death often resulted. On the other hand, no deaths were reported in cases where the current had passed between both hands or from the head to one or both hands. Killinger concluded that direct current is just as dangerous as alternating current when the current flows through the long axis of the body.

One of the great dangers of alternating current is its tetanizing effect on voluntary muscle. This is most pronounced in the frequency range of 15 to 150 c/s. When the muscles thus remain in spasm, a person can be "locked" to the contacts until the circuit is broken (Fig. 4). As the frequency increases, the danger becomes less and high frequency current of $^{1}/_{2}$ to 1 megacycles are harmless even at high voltages (Jaffe, 1928; Bryer, 1960; Kragh & Erich, 1961).

*5. Pathway of the current*

There is still no agreement about which structures the current follows through the body. Since the blood has low resistance many believe that the current follows the blood vessels (Lewis, 1958; Kragh & Erich, 1961 and many others). In Jaffe's (1928) opinion the current spreads radially into the body from the point of entrance, to be collected again at the point of exit. Weeks & Alex-

*Fig. 4.* This is reported to be the first picture in history of an electrical accident, while a Lord Major was introducing a Beauty Queen. Both hands locked to the microphones by tetanic spasm due to a short circuit. (From *Life Domestic,* March 2nd, 1953. By courtesy Photo Editor Orville Brunelle, The Lethbridge Herald, Alberta, Canada).

ander (1939) showed in animal experiments that the current traverses the body as if it were a structureless gel; always taking the shortest way between the points of contact, without any deflection by anatomical structures. Panse & Menden (1962) agree with this view.

Which ever structures the current may follow, it is certain that it can "leap" over the flexor surfaces of joints. In this context a practical important injury is described by Skoog (1963): When a patient grasps a high tension wire, the hand may be thrown into spastic flexion contraction and the current in taking the shortest distance, arcs across to enter at the wrist. The typical picture of this injury is a deep burn of the fingers and palm combined with major deep tissue destruction at the volar aspect of the wrist with sparing of the tissues in the region of the thenar eminence.

It has been stated that the danger to the heart is greatest when the current passes through the left arm to the feet (Jex-Blake, 1913; Schridde, 1926; Lewis, 1958). In contrast to this Freiberger showed that the current density in the heart was $1\,^1/_2$ to $2\,^1/_2$ times greater when the current entered the body through the *right* arm.

*6. Duration of the contact*

A prolonged contact will as a rule produce more severe damage than a short one. Extremely short exposures (which in German terminology are called 'Wischer') are usually harmless. Langworthy & Kouwenhoven (1931) showed that rats can survive shocks of 100 A at 200,000 V, provided the duration does not exceed 4 microseconds.

If a person is locked to the contacts for a prolonged period of time, he may succumb to a current which would otherwise not be lethal.

Clinical experience indicates that local lesions may occur in fractions of a second, even at low tension.

*7. Surface area of contact points*

The value of the current divided by the surface area, of the contact point is the current density. The smaller this area, the greater the current density and the more energy is transformed into heat. If the contact surfaces are large, the current density may not be sufficient to produce any local lesion at all, but as the skin is a bad insulator, the current flow may still be great enough to kill. The significance of the surface area of the contact points is best exemplified in low tension accidents: The child sucking a live extension cord sustains deep burns of the lip, but there are no systemic effects. A person lying in a bath tub touching an unearthed electrical element will be killed immediately, but local lesions are absent.

## THE MATERIAL

This study comprises 122 cases (Table 1) of varying severity, including out-patients from the vicinity of the hospital. Most of the severe cases were referred from other hospitals within an area comprising the middle and northern part of Sweden (Pop. ca 3,000,000). Patients referred with late sequelae of burns are also included in this material.

Table 1

| Patients referred for primary treatment | Hospitalized patients | Out patients |
|---|---|---|
| Time between injury and admission to Burns Unit | | |
| < 45 hours | 42 | 20 |
| 3–21 days | 18 | 5 |
| 22–180 days | 10 | 2 |
| > 180 days | 1 | 0 |
| Total of primary cases | 71 | 27 |
| Patients referred with sequelae of burns previously treated in other hospitals | 17 | 7 |
| Total all cases | 88 | 34 |

Of 789 burns treated in our unit, 7% were caused by electricity. A similar figure (8%) is reported from Stockholm by Hjern *& al.* (1966). These figures are high compared with those of foreign authors. Hay & Cronin (1954) give 2.8% Davies (1959) 3.7%, Blocker *& al.* (1961) 3.7% and Szczepanski (1964) 2.7%. The comparatively high figures in the Swedish series is probably due to the widespread use of electricity in this country.

The material will be considered as a whole when discussing general technical details directly associated with the accident. The clinical course in primary and secondary cases will be discussed separately.

### *Sex- and age distribution*

The vast majority (84%) of our patients are males. During early childhood the incidence of electrical injuries is almost the same in both sexes, but later male preponderance increases. Distribution according to sex and age among our cases is shown in figure 5, indicating a high incidence of electrical burns in small children. Most of these injuries were caused by biting inadequately insulated cables or sucking the free end of an extension cord. Another typical injury in children is burns of the fingers resulting from touching the live contacts of a wall outlet. Finally, another contributing factor to the high incidence of electrical injuries in childhood is the temptation to

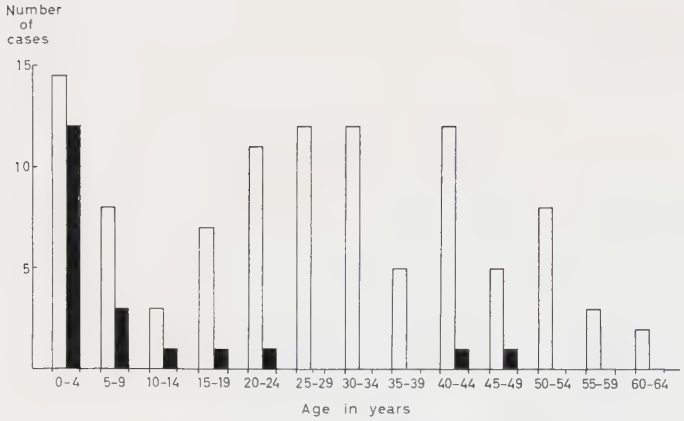



*Fig. 5.* Sex—and age distribution of 122 patients with electrical burns. Black columns females.

play in forbidden areas such as railway stations, building sites etc.

Young adults seem to be more accident prone than older ones. An age distribution similar to ours appears in fig. 6 which represents injuries to line workers and engineers employed in SJ (National State Railways). A possible explanation of this is that the majority of burns in adults are occupational accidents which tend to affect the younger and less experienced workers. The lower incidence in the older age groups may be attributed to broader experience and caution. Furthermore, older workers are often transferred to less dangerous jobs.

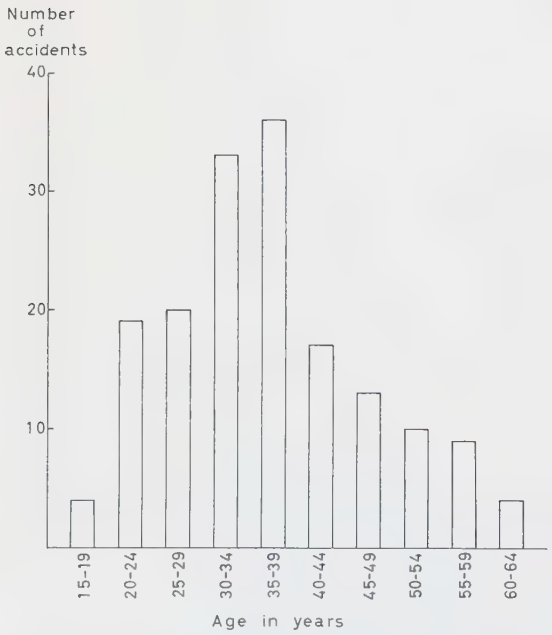



*Fig. 6.* Age distribution of line workers and engineers injured by electricity during the period 1951 to 1961. (National State Railways.)

*Place of accident*

The total number of occupational injuries in this material is 67 (55%) compared to 41 (34%) domestic injuries. 14 patients (11%) were injured elsewhere. In adults (≥15 years) occupational injuries constitute 83% of all cases while only 9% were injured in or around the home. The most severe burns in our series and those which presented the greatest therapeutic problems were the ones resulting from occupational accidents, largely because these tended to be high tension injuries.

*Monthly distribution of accidents*

Electrical accidents resulting in burns were more frequent during the spring and summer months. Figure 7 A shows, however, that the seasonal difference is due entirely to the variation in occupational accidents. Figure 7 B shows the monthly distribution of accidents in the National State Railways. The higher frequency in the summermonths is indisputable in our series. It should be mentioned however that much of the work on high tension installations such as painting etc. is typical out door work, which owing to the Swedish climate is almost exclusively carried out during the summer. Furthermore, these burns are often more extensive because less protective clothing is used during the warmer months. A definite maximum during the summermonths has also been reported by Jaeger (1921) and François & Cabanes (1962).

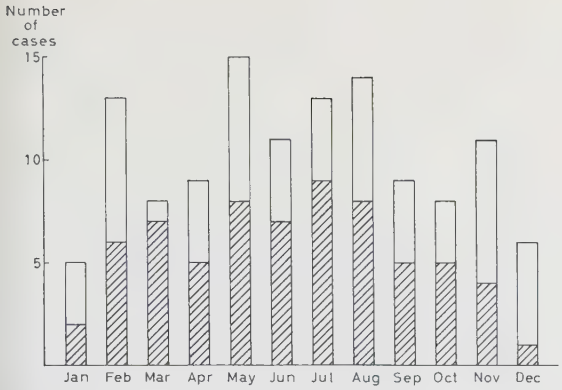



*Fig. 7 A.* Monthly distribution of accidents in 122 patients with electrical burns. Hatched columns—occupational injuries.

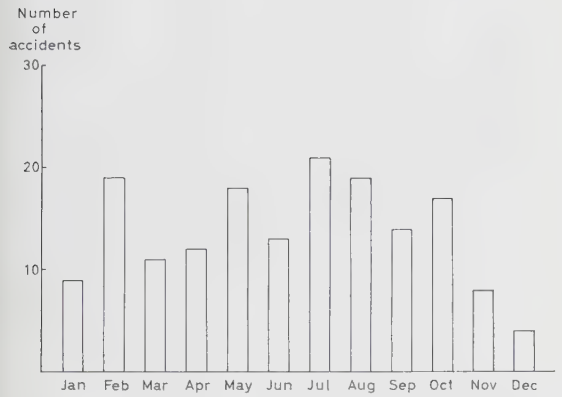



*Fig. 7 B.* Monthly distribution of accidents during the period 1951 to 1961 in line workers and engineers in The National State Railways.

### *Voltage and type of current*

The patients had been exposed to electricity at potentials from 6 to 70,000 volts. Of these 57% were injuries by high tension (⩾380 )V (Table 2). It was characteristic that burns caused by currents at 380 V or more, were deeper and more extensive than those caused by low tension, thus protracting treatment time and incapacity for work. If a person was injured when touching one outside wire and the neutral in a 380 V system he was classified in the low tension group.

In the low tension group the voltage was never less than 127 V, except in four cases where persons got burned when changing car batteries. Sinces these batteries have a small electromotive force (6–12 V) they are generally harmless, but when fully charged a battery of this type can deliver a short circuit current of several hundred amperes. One of the patients injured by the current from a car battery had his wedding ring melted away, leaving a circular burn on his finger, and in another patient the links of a wrist-watch were fused.

Table 2. *Distribution of accidents according to the voltage*

Fatal cases within brackets.

| | Number of cases | |
|---|---|---|
| | Low tension <380 V | High tension ⩾380 V |
| At work | 8 | 59 (5) |
| At home | 39 | 2 |
| Elsewhere | 7 | 7 |
| Total | 54 | 68 (5) |

Twelve patients were injured by direct current at potentials from 6 to 40,000 V. 110 patients were injured by alternating current, the frequency being from 16 $^{2}/_{3}$ to 50 c/s. No observations were made in our material to prove that burns from direct current were more severe, as have been stated by Uzac (1936), Dale (1954), and Hyslop & Miller (1956).

No attempt was made to estimate the rate of current flow through the patient during the accident.

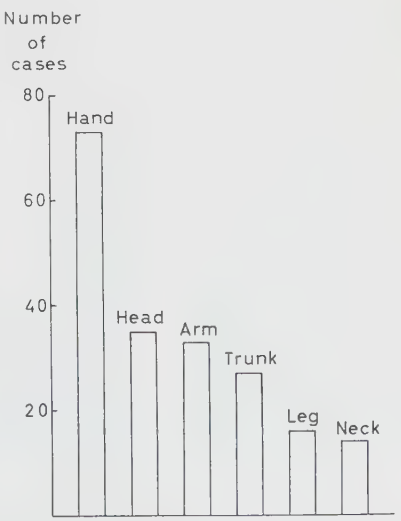



*Fig. 8.* Location of burns in 98 patients referred for primary treatment.

## PATIENTS REFERRED FOR PRIMARY TREATMENT

*Location of the burns*

The location of the electrical lesions in the present series is shown in figure 8. As in other burns the hands were the most common site of injury. Out of 98 cases referred for primary treatment 73 persons had burns localized to one or both hands. It was found that when burns were localized to the hands alone, they were in 65% caused by low tension. In high tension accidents the hand lesions were frequently combined with remote tissue destruction due to arcing of the current.

Burns localized to the head were of varying severity, from superficial arc burns of the face to deep destruction extending into the bone. Full thickness injuries of lips and eyelids were also included.

*Type of burns*

Low tension burns were almost exclusively of the contact type, lesions due to arcing being extremely rare (Table 3). Burns were usually found at both contact points although in some cases they were demonstrable at one point only. Burning did not occur where one contact point was of larger area (thus decreasing the current density) than the other or where the skin was moist. The most characteristic local lesions encountered in the low tension group were small, deep burns (Fig. 9), which sometimes involved vessels, tendons and nerves. These burns were sharply defined and surrounded by an ischaemic zone and a slightly hyperaemic halo. Because of their depth they were insensible to touch, but despite this, patients in many instances complained of pain which apparently was of neurogenic origin. Occasionally the so called "current marks" were found at the contact points (Fig. 9). These typical lesions originally described by Jellinek (1922) are usually circular or oval in shape, the diameter varying between a few millimeters and several centimeters. The area is often slightly elevated with a central crater-like depression. The colour is white, grey or yellowish grey. The nature of the current mark has been much in dispute (Schridde, 1922; Jellinek, 1923, 1960; Koeppen & Gerstner, 1935; Dale, 1954; Fornari & Mazzucchelli, 1964), but most authors seem to agree that the basic pathology is a coagulation necrosis, caused by the transformation of electrical energy into heat during its passage through the tissues. The mechanism is the same as the formation of a contact burn. According to Jellinek (1923, 1954) current marks are painless and never become infected. In our series, patients were not routinely questioned about pain, but some definitely mentioned that they had pain from lesions which were obviously current marks. All current marks in our patients healed spontaneously without infection, leaving insignificant scars.

Table 3. *Type of burns in relation to the tension*

Fatal cases within brackets.

| | Number of cases | |
|---|---|---|
| | Low tension | High tension |
| Contact burns | 34 | 22 |
| Arc burns | 1 | 22 (2) |
| Mixed burns | 0 | 19 (3) |
| Total | 35 | 63 (5) |

*Fig. 9.* Case 8. Low tension burn of left ringfinger. Note the typical small current mark on the middle finger.

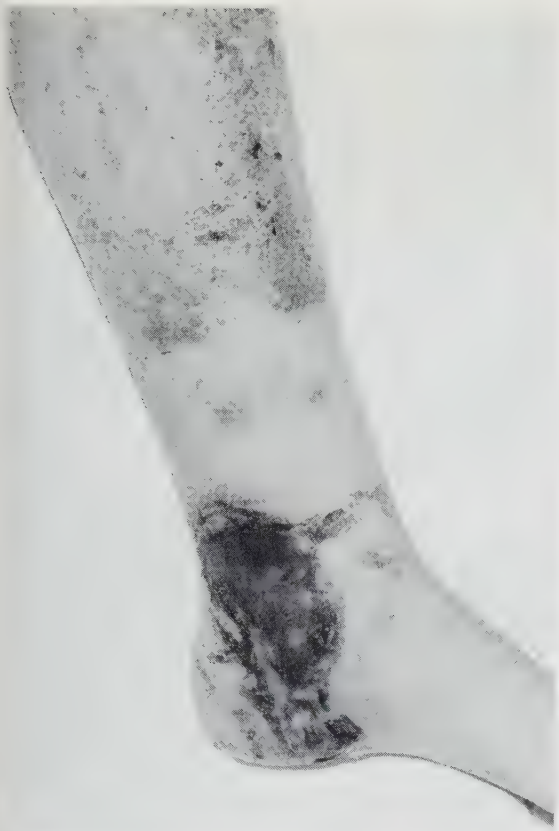


*Fig. 10.* Case 71. High tension burn in a boy, aged 11. A current at 16,000 V arced across to his head and neck and earthed through both feet. Photo of exit point 23 days after injury.

In the high tension group the number of contact burns, arc burns and mixed burns were fairly even (Table 3). In general, high tension contact burns were similar to those produced by low tension, except that they were larger and deeper (fig. 10). In many cases the typical picture of contact burns was obscured by additional arc or flame burns.

Arcing is by no means a phenomenon entirely limited to the highest voltages but may occur with disastrous results even at 380 V, depending upon the power. Eleven patients sustained burns of varying severity from arcing in 380 V installations. Because of the extremely high temperature in the arc, volatilized metal particles from the electrodes can be impregnated into the skin by the blast and give rise to a peculiar metallic appearance (Fig. 11). Such metallization is usually harmless and disappears gradually with separation of the crusts. In general, the burns produced by arcing did not differ from ordinary thermal burns. The arc often ignited clothing, thereby producing additional flame burns.

*Severity of the burns*

The severity of the burns in our series was studied from various points of view. In patients admitted for primary treatment the extent of the burned area was estimated according to the Berkow

*Fig. 11.* Case 96. Arc burn and metallization in an electrician, aged 20. The temperature of the arc volatilized the copper contacts and the blast impregnated the face with copper particles. (A) Condition on admission to the Burns Unit 11 days after injury. The face had a brownish-grey metallic appearance. (B and C) 3 months after injury. No traces of metal particles. Facial burns healed spontaneously.

*Fig. 12.* Case 73. High tension burn (380 V) in a crane operator, aged 23. (A, B) Condition on admission 7 weeks after injury. When necrotic portions of the left malar and frontal bones were removed a large epidural abscess was found over the frontal cortex. The major portion of the defect was covered by an acromiopectoral tube pedicle, supplemented posteriorly by a transposition flap of scalp and post-auricular skin. Bone grafts were added later to fill out the defect in the frontal bone. (C) 10 weeks after injury. The necrotic and fibrosed tissue excised from the right side of the face and neck included the angle of the mandible. This relieved his trismus, which was quite severe. Closure by direct approximation, the scar being improved by later Z-plasties. (D, E and F) Final result 3 years later. This patient developed epilepsy 2 years after injury; EEG showed an epileptogenic focus within the frontal lobe.

method. It was then found that in 26 patients out of 71 the area involved was less than 1%, compared with 46 patients out of 70 in a similar series reported from Birmingham (Davies, 1959). In a series of 54 cases treated in the University Hospital of Stockholm, Hjern *& al* (1966) found that 19 patients had burns involving less than 2% of the body surface. Of these 15 were injured by low tension and 4 by high tension. A comparison between these three series is shown in table 4. The reason for the discrepancy between the English and the Swedish series is probably that the majority of the Swedish cases (68% in our series) represent occupational injuries—almost exclusively caused by high tension—compared with 46% injured at work in the Birmingham series.

To evaluate the severity, the depth of the burns must also be considered, and for that reason our

Table 4. *Extent of burned area in patients admitted for primary treatment*

B = Birmingham series, S = Stockholm series (high tension burns only), U = Uppsala series (for comparison out patients are omitted).

| Total extent of body surface involved in burns, % | Number of patients | | |
|---|---|---|---|
| | B | S | U |
| < 1 | 46 | — | 26 |
| 1– 9 | 20 | 13 | 20 |
| 10–19 | 3 | 5 | 7 |
| 20–29 | 0 | 5 | 8 |
| 30–39 | 0 | 5 | 6 |
| 40–49 | 1 | 4 | 2 |
| 50–59 | 0 | 1 | 1 |
| 60–69 | 0 | 2 | 1 |
| 70–79 | 0 | 1 | 0 |
| 100 | 0 | 1 | 0 |
| Total | 70 | 37 | 71 |

material has been classified into three groups of severity according to Barnes (1957).

| Severity group | 1st–2nd degree | 3rd degree |
|---|---|---|
| I | < 15 % | < 5 % |
| II | 15–30 % | 5–15 % |
| III | > 30 % | > 15 % |

This classification is particularly useful in surface burns and is therefore mainly applicable to arc burns where the effect of the injury depends more on the surface area involved than on the depth. It does not however give a true picture of the severity of contact burns where relatively small but deep lesions may be of the highest degree of severity (Fig. 12).

A total of 34 low tension burns were classified in severity group I. The severity of 64 high tension burns is shown in table 5.

Table 5. *Severity of high tension burns in 63 patients referred for primary repair*

Fatal cases within brackets.

| | Severity groups | | |
|---|---|---|---|
| | I | II | III |
| Contact burns | 20 | 2 | 0 |
| Arc burns | 17 | 3 | 2 (2) |
| Mixed burns | 3 | 11 | 5 (3) |
| Total | 40 | 16 | 7 (5) |

*Significance of the pathway of the current*

Most authors agree that the pathway of the current through the body is important in relation to its general effects but few attempts have been made to correlate the pathway to the severity of the local lesions. Brown & Moritz (1964) classified their cases according to the current pathway but were mainly concerned with the systemic effects of each pathway. Hjern & *al.* (1966) found that current flow through one hand to the feet was associated with the most severe peripheral injuries, often resulting in amputations.

In order to evaluate the significance of the current path our material has been divided into 6 groups (A–F) according to the resistance. The relative resistance values for these groups should be regarded only as approximate. This classification is mainly based on the studies of Freiberger and Osypka as shown in fig. 3, page 8 and the groups are listed in decreasing order of resistance:

A: Hand → hand, hand → foot or vice versa. (Two resistors in series. Total resistance arbitrarily set at 100%.)

B: Hand → both feet, foot → both hands or vice versa. (One resistor in series with two resistors in parallel. Total resistance 75%).

C: Both hands → both feet or vice versa. (Two pairs of resistors in parallel, coupled in series. Total resistance 50%.)

D: Hand → trunk, foot → trunk or vice versa. Or the current enters and leaves the same extremity. (One resistor in series. Total resistance 50%.)

E: Both hands → trunk, both feet → trunk or vice versa. (Two resistors in parallel. Total resistance 25%.)

F: Current flow through the head, regardless of the current flow through other parts of the body. It is possible that the resistance of the head may be of the same magnitude as that of an extremity, but because of the effects upon the brain, all cases with current flow through the head were separated in one group.

When discussing the importance of the current pathway, one must distinguish between the systemic effects and injury to internal organs on one side, and the local lesions on the other side. In this context the term local lesion implies both the burn at the contact point and the extension of

Table 6. *Number of patients rendered unconscious by high tension shocks*

| | Number of patients |
|---|---|
| Pathway | |
| A | 10 |
| B | 3 |
| C | 1 |
| D | 1 |
| E | 2 |
| F | 11 |

this lesion into the deep tissues of an extremity where the current density is greatest and the resistance highest.

SYSTEMIC EFFECTS AND DAMAGE TO INTERNAL ORGANS

*Low tension*

In the low tension group 7 patients became locked to the contacts due to the tetanic muscular contraction caused by alternating current. This happened twice in pathway A (hand→hand) and five times in pathway D (In–out the same extremity). The duration of adhesion to the contacts, as reported by the patients was uncertain, but the clinical signs generally indicated a short duration since the lesions were slight in most cases.

In one case, where the current flow was said to have been through the left hand and arm, the patient was rendered unconscious for 10 minutes. Although this may be a factual account of what happened, unconsciousness induced by peripheral current flow is very rare in low tension accidents (Panse, 1955). The patient who was 54 years old, had previously been treated for hypertension and an ordinary syncope provoked by the psychic shock is perhaps a more plausible explanation. The duration of unconsciousness on the other hand is very suggestive of current flow through the head, and this may have taken place without the patient being aware of it. None of the patients in the low tension group showed signs of damage to the heart and electrocardiographic examination was performed in only four cases. They were all normal. There were no systemic effects suggestive of damage to other viscera.

*High tension*

In high tension accidents, locking to the contact was uncommon. Only 3 patients in pathway A and 2 patients in pathway D were locked to the contacts for a few seconds. Some of the patients were thrown away from the contacts and some fell. This infrequency of locking, with higher voltages is probably explained by the fact that the circuit is completed by arcing before the patient touches the contact.

A more characteristic feature in the high tension group was neurological symptoms. 67% of the patients with contact burns were rendered unconscious and this happened to every person in whom the current passed through the head. It should be noted that unconsciousness also occured in 17 patients where the current had not traversed the head. (Table 6.) All unconscious patients—except two fatal ones—recovered within a few hours, the majority regaining consciousness in 5–10 minutes. In four cases (Pathway A, D, E and F) the unconsciousness might have been caused by falling.

Transitory convulsions resembling grand mal were observed in two patients (Pathway A and D), Agitation and confusion were reported once with pathway A, once with E and twice with F. All these patients recovered quickly.

For practical reasons EEG examinations were not carried out routinely and only 3 patients underwent EEG examination in the acute phase. The test was normal in each case.

Other immediate effects upon the nervous system produced a variety of symptoms ranging from paresthesiae to pareses. It is doubtful whether symptoms from peripheral nerves should be considered under this heading, since their injury properly should be regarded as part of the local lesion. Indeed similar symptoms may be caused by lesions of the spinal cord. These problems will therefore be subjected to closer analysis in a separate chapter.

Disturbances in heart function were observed only twice. In a boy, aged 11 the current arced across to his head from a 16,000 V live wire while he was playing on an engine at a railway station. The current earthed through both feet (Pathway F). On admission to the Burns Unit 5 hours later ECG examination revealed a partial intraventricular block which persisted for 10 days. The other patient, an engineer aged 47, was injured by a 6000 Volt transformer. The current entered through the forehead region and praecordium and earthed through both hands

Table 7. *Current Pathway A: Current flow hand→hand*

| Case number | Age years | Location of burns | No opns. | Graft opns. | Tot. hosp. time (days) | Healing time (days) | Complications | Incapacity for work (days) | Rated disability |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 13 | Both hands | — | — | — | 30 | — | — | — |
| 2 | 40 | Both hands | — | — | — | 14 | — | — | — |
| 3 | 6 | Both hands | 12 | 8 | 111 | 99 | — | — | — |
| 4 | 2 | Both hands | 2 | — | 36 | 36 | Amp. r. 2nd, 3rd finger | — | not yet decided |
| 5 | 1 | Both hands | 5 | — | 36 | 24 | Amp. r. 5th finger | — | not yet decided |
| 6 | 1 | R. hand | 3 | 3 | 54 | 45 | Amp. r. 4th finger | — | not yet decided |
| 7 | 6 mos. | Both hands | 1 | 1 | 24 | 24 | | — | — |
| 8 | 52 | L. hand | 1 | 1 | 3 | 52 | | 30 | — |
| 9 | 24 | R. hand | 2 | 2 | 6 | 72 | | 72 | — |
| 10 | 33 | Both hands | 1 | 1 | 1 | 45 | | 49 | — |
| 11 | 3 | Both hands | 3 | 2 | 33 | 30 | | | |
| Average | 10 | | 3.3 | 2.6 | 34 | 43 | | 50 | |

(Pathway F). After 5 minutes of unconsciousness he became confused and agitated and on admission to the Burns Unit, bradycardia (pulse 55 min) and nodal rhythm were observed. The pulse rate became normal within 24 hours and one week later ECG was also normal.

Two patients had myocardial changes apparently unrelated to their injury. In 12 patients, ECG was normal on admission to the Burns Unit. ECG examinations were not performed on all patients with high tension burns, because the site and type of the lesions did not always permit proper placing of the leads.

Other systemic effects were not observed, nor any immediate tissue damage to internal organs, apart from one case where the current caused coagulation of brain tissue.

## LOCAL LESIONS

### *Low tension burns*

About 50% of the patients with low tension burns were children under 10 years of age. The main cause of injury was playing with inadequately insulated extension cords or lamp sockets.

A common feature of low tension contact burns was that they were almost exclusively localized to the hands or the mouth. Although the burns were often deep, the extent was always less than 1% and in the majority of cases their only significance was that they showed current passage had taken place. The lesions were deeper in patients who had been locked to the contacts. The current pathway could be ascertained in every case, and on this basis the cases were separated in two major groups, which presented different characteristics.

### *Pathway A*

This group comprises 11 patients (Table 7) who were all traversed by the current between both hands. The burns were generally bilateral but in 3 cases were unilateral. In bilateral lesions the burns were usually deep on one side and rather superficial on the other, depending on the resistance and surface of the contact. One could not distinguish between entrance and exit wounds by the appearance of the burn. In one case the patient had a firm grasp with his right hand on a metal ladder, when he accidentally touched a small live contact with his left hand. The current earthed through his right hand, but burns were only present at the entrance point.

Although these burns, because of their small area could never be classified as severe, they were always 3rd degree and 9 patients required hospitalization. In 3 children (cases 4, 5, 6) partial amputation of one or more fingers had to be performed, and skin grafting was necessary in 5 cases. Split skin grafts were used for primary repair in 2 patients (cases 6, 11) and full thickness skin in 2 others (cases 8, 9). In case 3 the flexor tendons of the middle finger were exposed and an abdominal flap was transferred after excision of necrotic tissues.

Table 8. *Current Pathway D: Current enters and leaves through one hand or arm*

| Case number | Age years | Location of burns | No opns. | Graft opns. | Tot. hosp. time (days) | Healing time (days) | Incapacity for work (days) |
|---|---|---|---|---|---|---|---|
| 12 | 4 | R. hand | — | — | — | 43 | — |
| 13 | 5 | R. elbow | — | — | — | 27 | — |
| 14 | 30 | L. hand | — | — | — | ? | ? |
| 15 | 8 | R. hand | — | — | — | ? | — |
| 16 | 53 | R. hand | — | — | — | ? | ? |
| 17 | 16 | L. hand | — | — | — | 21 | 5 |
| 18 | 49 | R. hand | — | — | — | 90 | 103 |
| 19 | 30 | R. hand | — | — | — | ? | ? |
| 20 | 19 | L. hand | — | — | — | 21 | 24 |
| 21 | 16 | L. wrist | — | — | — | 14 | 14 |
| 22 | 23 | L. hand | — | — | — | 12 | 15 |
| 23 | 3 | L. hand | — | — | — | 16 | — |
| 24 | 5 | L. hand | — | — | — | 13 | — |
| 25 | 32 | ? hand | — | — | — | ? | — |
| 26 | 7 | L. hand | — | — | — | 30 | — |
| 27 | 54 | L. hand | — | — | 5 | 62 | 153 |
| 28 | 2 | R. elbow | 2 | 1 | 15 | 35 | — |
| 29 | 12 | R. hand | 1 | 1 | 4 | 36 | — |
| 30 | 8 | L. hand | 5 | 4 | 69 | 62 | — |
| 31 | 9 | L. hand | 2 | 1 | 4 | 42 | — |
| Average | 19 | | 2.5 | 1.8 | 19 | 35 | 52 |

Secondary scar excision and skin grafting was carried out in 7 patients, including cases 6 and 11 where the split grafts were replaced by full thickness ones.

Average hospitalization time for the 9 patients admitted for primary repair was 30 days, and the average healing time for all (including 2 out-patients) was 36 days. The incapacity for work averaged 50 days for 3 patients. One female adult was able to continue with her work during the period of treatment. The rest of the patients were children.

*Late results.* All 11 patients reported for personal examination. The average observation time in this series was 6.5 years (range $^1/_2$–13). Sequelae were observed in 3 patients (cases 4, 5, 6) where amputation of fingers had been performed, but apart from that there was no disability. The other patients were completely restored and the grafts had all good quality with slight impairment of sensation.

*Pathway D*

In this group (Table 8) comprising 20 patients, current pathway was short, with entrance and exit points being on the same limb. It was also characteristic that the burns in many of these cases were superficial. Out of 20 patients 8 had burns of only 2nd degree, while the rest had 3rd degree burns. Transient loss of finger sensation was observed in 2 patients (cases 26, 29) but there were no other injuries to deep structures.

In 5 patients (cases 27–31) hospitalization was necessary. In one patient (case 27) spontaneous healing took place. Split skin grafts were used for primary repair in 3 patients (cases 28, 29, 30) and in one (case 31) a local flap was used to cover a relatively deep burn of the left index finger. Secondary full thickness skin grafting and scar correction was performed in one patient (case 30) on four occasions.

Average hospitalization time for these patients admitted for primary repair was 15 days. The average healing time for 15 patients (including 10 out-patients) was 35 days. Five out-patients did not return after the first dressing and the healing time in these cases could not be ascertained. As judged by the lesions however, a healing time of 10–12 days seems reasonable. This gives an estimated healing time of less than 30 days for the whole group.

Average incapacity for work was 52 days for 6 patients. It should be mentioned however that these were all treated conservatively and in 2 patients (cases 18, 27) the incapacity was 103 and

Table 9. *Special Pathway*

| Case number | Age years | Location of burns | No opns. | Graft opns. | Tot. hosp. time (days) | Healing time (days) | Complications |
|---|---|---|---|---|---|---|---|
| 32 | 4 | L. lip | — | — | — | ? | Haemorrhage |
| 33 | 6 mos. | U. lip | 2 | 1 | 7 | 30 | Haemorrhage |
| 34 | 1 | L. lip | 1 | — | 5 | 36 | — |
| Average | 1.8 | | 1.5 | — | 6 | 33 | — |

153 days respectively. It is highly probable that this time could have been reduced considerably by early surgical treatment. In 2 cases the time of incapacity for work was unknown, but it was probably not more than 21 days. 2 out-patients were able to continue with their work during the period of treatment. The rest of the patients were children.

*Late results.* Out of 20 patients, 13 reported for examination and 3 answered a questionnaire. One patient had died from cardiac insufficiency due to aortic stenosis 3 years after the accident, and 3 patients could not be traced. The average observation time was 4.6 years (range 2 months–15 years).

All patients stated that they were completely recovered and no sequelae were observed in the patients who were examined. The grafts were slightly hyperpigmented, but of good texture and with normal sensation. Sensation was normal in the fingers of cases 26, 29. Movement was unrestricted in all affected limbs, but in one patient (case 26) correction of a minor scar in the thumb web was required.

There is reason to believe that recovery was complete in the 4 cases not followed up, as their lesions were expected to heal spontaneously within a short time.

*Comment.* In current pathway A the resistance is considerably higher than in current pathway D owing to the short distance of the latter.

The most severe local lesions would be expected where the current had passed through pathways of high resistance. This was confirmed by the clinical findings. Burns associated with current pathway A were generally deeper, more often required hospitalization and surgical treatment, and had a longer healing time than those associated with current pathway D.

*Special pathway*

Three children (cases 32, 33, 34) were admitted on account of full thickness burns of the lips (Table 9). Two of them (32, 33) were admitted as emergencies on account of severe secondary haemorrhage on the 9th and 17th day respectively. On admission haemoglobin was 57% in both cases. In one of these patients (case 33) the superior labial artery was ligated and primary excision with mucosal flap repair carried out. This patient was later admitted for secondary correction on two occasions. In the other case, the haemorrhage had subsided on admission. He was treated conservatively and later referred to a plastic surgical unit near his home. In the third case excision and closure was performed when the patient was referred 4 weeks after the accident. In this case no arterial bleeding was observed.

*Late results.* Two patients (cases 33, 34) have reported for examination after 4 and 9 years respectively. Both were well healed with good function. The cosmetic result was acceptable but further correction was recommended in both cases. The parents however were satisfied and further surgery was postponed. Development of the mandible and maxilla was normal in both cases, as also were speech and dentition. Case 32 could not be traced.

*Comment.* In this type of lesion the current pathway is not more than 1.5 cm and the resistance of the richly vascularized lower lip (which is the predilection site) is probably small. It is therefore unlikely that only the passage of the current could generate sufficient heat to cause such deep burns. The deep destruction frequently encountered in these cases may be explained by superimposed arc burns. Arcing may occur in two different ways: Sucking the free end of a live ex-

Table 10.

| Case number | Age years | Tension (volt) | Location of burns | Tot. area of burns (%) | No opns. | Graft opns. | Tot. hosp. time (days) | Complications | Healing time (days) | Incapacity for work (days) | Rated disability (%) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *Current Pathway A: Current flow hand→hand or hand→foot* | | | | | | | | | | | |
| 36 | 19 | 650 | L. hand | 1 | — | — | — | — | 21 | — | — |
| 37 | 40 | 380 | L. hand | 1 | 1 | — | — | Part. amp. l. thumb | 87 | 87 | — |
| 38 | 49 | 20,000 | Hands, arms, head, trunk | 22 | 9 | 4 | 168 | Amp. l. 5th finger Nerve lesion | 106 | 1095 | 25 |
| 39 | 36 | 40,000 | Head, arms, foot | 2 | 6 | 6 | 88 | Amp. r. 1st, 2nd, 3rd toes | 102 | 199 | — |
| 40 | 27 | 4,000 D.C. | Hands | 1 | 2 | 2 | 5 | — | 47 | 141 | — |
| 41 | 50 | 70,000 | Trunk, arms | 15 | 2 | 1 | 87 | Renal failure | 103 | 365 | 15 |
| 42 | 34 | 380 | Hands | 1 | 4 | 4 | 23 | — | 97 | 260 | — |
| 43 | 22 | 10,000 | Hands, arms, head, trunk | 15 | 20 | 10 | 160 | Amp. r. lower arm & l. 2nd, 3rd, 4th, 5th fingers | 155 | 893 | 100 |
| 44 | 34 | 10,000 | Hands, arms, head, trunk, leg | 20 | 17 | 12 | 218 | Amp. r. lower arm | 163 | 1095 | 70 |
| 45 | 10 | 16,000 | Hands, arms, head, trunk | 30 | 3 | 2 | 41 | — | 45 | — | — |
| 46 | 53 | 70,000 | L. arm, trunk, r. leg | 25 | 29 | 19 | 432 | Drop foot, Parkinsonism | 81 | 730 | Not yet decided |
| 47 | 40 | 6,000 | Hands, arms, leg | 6 | 4 | 2 | 38 | — | 58 | 30 | — |
| 48 | 36 | 500 | Hands, trunk | 2 | 11 | 6 | 75 | Amp. l. thumb | 59 | Still under treatment | Not yet decided |
| 49 | 25 | 16,000 | Hands, arms, trunk | 30 | 2 | 1 | 47 | — | 59 | Still under treatment | Probably none |
| Average | 34 | | | | 8.5 | 5.8 | 115 | | 85 | 490 | |
| *Current Pathway B: Current flow hand→feet, hands→foot* | | | | | | | | | | | |
| 50 | 45 | 3,000 | Hand, feet | 1 | 3 | 1 | 72 | Tetanus | 72 | 300 | — |
| 51 | 51 | 1,000 | Arms, trunk, foot | 6 | 6 | 3 | 90 | — | 103 | 240 | 33 |
| Average | 48 | | | | 4.5 | 2 | 81 | | 88 | 270 | |
| *Current Pathway C: Current flow both hands→both feet* | | | | | | | | | | | |
| 52 | 16 | 16,000 | Hands, feet | 5 | 12 | 3 | 117 | Amp. r. 1st, 2nd toes. Nerve lesion | 129 | 485 | 40 |
| 53 | 28 | 70,000 | Hand, arm, trunk, feet | 10 | 7 | 4 | 50 | Part. amp. r. 1st, 2nd toes | 105 | 110 | — |
| Average | 22 | | | | 8.5 | 3.5 | 84 | | 117 | 298 | |

*Current Pathway D: Current enters and leaves through the same hand or arm*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 54 | 29 | 380 | L. hand | 1 | — | — | — | — | 12 | — | — |
| 55 | 44 | 500 | L. hand | 1 | 1 | — | 3 | — | 40 | 42 | — |
| 56 | 31 | 16,000 | L. arm, trunk | 20 | 3 | 2 | 46 | — | 45 | 85 | — |
| 57 | 2 | 380 | L. hand | 1 | 2 | — | 18 | Part. amp. l. index finger | 72 | — | — |
| 58 | 9 | 16,000 | L. arm, trunk | 20 | 8 | 6 | 152 | — | 61 | — | — |
| 59 | 2 | 380 | R. hand | 1 | 1 | — | 50 | — | 61 | — | — |
| 60 | 34 | 16,000 | Arms, trunk | 25 | 2 | 1 | 55 | Paralysis l. recurrent nerve | 102 | 177 | — |
| 61 | 58 | 6,000 | R. arm, trunk | 1 | — | — | 3 | — | 14 | 13 | — |
| 62 | 41 | 380 | L. hand, arm | 1 | 2 | 2 | 32 | — | 72 | 80 | — |
| Average | 28 | | | | 2.8 | 2.8 | 45 | | 54 | 79 | |

*Current Pathway E: Current flow hands → trunk or feet → trunk*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| 63 | 50 | 1,700 | Trunk | 1 | 1 | — | 4 | Fract. Th. 6 | 38 | 68 | — |
| 64 | 40 | 16,000 | Hands, trunk | 20 | 3 | 2 | 46 | Hypoacusis | 95 | 160 | 15 |
| 65 | 44 | 16,000 | Head, trunk, u. & l. extr. | 60 | — | — | 16 | Sepsis † | — | — | — |
| Average | 45 | | | | 1.3 | 2 | 22 | | 67 | 114 | |

*Current Pathway F: Current flow through the head*

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 66 | 47 | 6,000 | Head, trunk, r. hand | 5 | 2 | 1 | 41 | Cataract | 81 | 153 | — |
| 67 | 26 | 380 | Head, l. arm | 1 | 1 | 1 | 10 | | 24 | 41 | — |
| 68 | 17 | 6,000 | Head, r. arm | 2 | 2 | 2 | 37 | — | 77 | 100 | — |
| 69 | 28 | 70,000 | Head, trunk, u. & l. extr. | 35 | 7 | 3 | 66 | — | 78 | 163 | — |
| 70 | 42 | 380 | Head | 1 | 4 | 2 | 60 | — | 121 | 139 | — |
| 71 | 11 | 16,000 | Head, trunk, feet | 28 | 2 | 1 | 53 | Lesion of the cord | Still | under | treatment |
| 72 | 21 | 3,800 | Head, hands | 2 | 11 | 7 | 139 | Nerve lesion | 130 | 1412 | 35 |
| 73 | 25 | 380 | Head | 2 | 19 | 25 | 403 | Epidural abscess. Loss of l. eye | 265 | 584 | 66 |
| 74 | 35 | 16,000 | Head, trunk, arms | 40 | 26 | 20 | 330 | Ectropium. Mental troubles | 104 | 1335 | 40 |
| 75 | 22 | 16,000 | Head, neck, trunk, legs | 40 | — | — | 6 | Brain contusion, liver rupture † | — | — | — |
| 76 | 38 | 16,000 | Head, neck, trunk, u. & l. extr. | 33 | 1 | — | 20 | Sepsis † | — | — | — |
| Average | 28 | | | | 7.6 | 5.8 | 108 | | 110 | 486 | |

†: patient died.

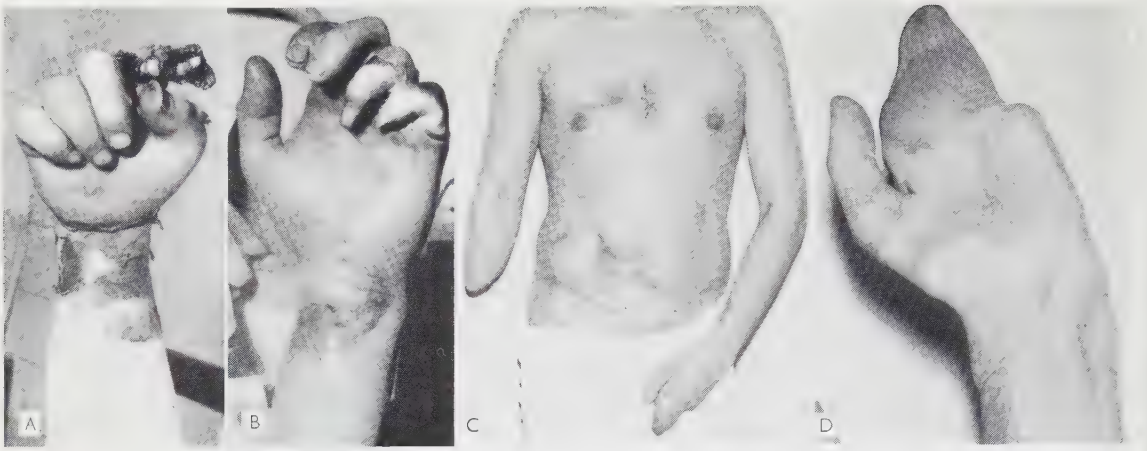



*Fig. 13.* High tension burns in an electrician, aged 25. A current at 10,000 V passed between both hands. (A and B) Condition shortly after injury. Note sparing of thenar and hypothenar emminences. (C and D) 7 years after injury. Note the dark appearance of the abdominal pedicle graft and atrophy of thenar and hypothenar regions. Adherent scars in both antecubital fossae after "leaping" of the current.

tension cord tends to produce a short circuit between the contacts as saliva is a good conductor. Alternatively, chewing through the insulating material of a live cord may result in a direct "short" between the two wires. In either case the heat generated in the arc is great enough to cause deep burns. According to Kazanjian & Converse (1958) electrical burns of the lips should be regarded as arc burns.

Animal experiments indicate that arterial changes in the vicinity of electrical burns are due to heat rather than the specific action of the electrical current (Jaffe *& al.* 1929). The most sensitive part of the vessel wall is, according to these authors, the media. Damage to the muscle fibres allows the vessel to become dilated and spontaneous rupture may easily occur. Progressive necrosis (Muir, 1958) resulting from the inevitable supervening infection may also contribute to this complication. Owing to its superficial course the labial artery is susceptible to such damage and the possibility of secondary haemorrhage in burns of the lips should always be kept in mind. Patients with this type of injury ought to be hospitalized for early surgical treatment or until separation is complete and healing is in progress.

*Arcing*

One patient who sustained superficial low tension arc burns of the face was treated as an out-patient and stayed home from work for 2 days. On follow-up 11 years later he was completely recovered.

*High tension burns*

The current pathway was sometimes difficult to ascertain in high tension burns, on account of the contact burns being obscured by additional arc and flame burns. A further confusing factor was that contact burns were occasionally absent at the entrance point of the current, although exit marks were always present. This phenomenon was especially observed in the highest voltage injuries where the current arced across to the patient as soon as he came within sparking distance of the live wire. Thus the current entered the patient over a relatively large surface, producing lesions of the arc burn type, while earthing through one or both feet was associated with typical contact burns at the exit points. However in most cases the current pathway was easy to ascertain. In doubtful cases a careful history together with the clinical signs, implemented by reports from electrotechnical experts usually resulted in a reasonable reconstruction of the current pathway.

*Pathway A*

This is the largest group, comprising 14 patients (Table 10). Out of these, 10 were traversed by the current between both hands and bilateral lesions were always found. In 4 patients the current

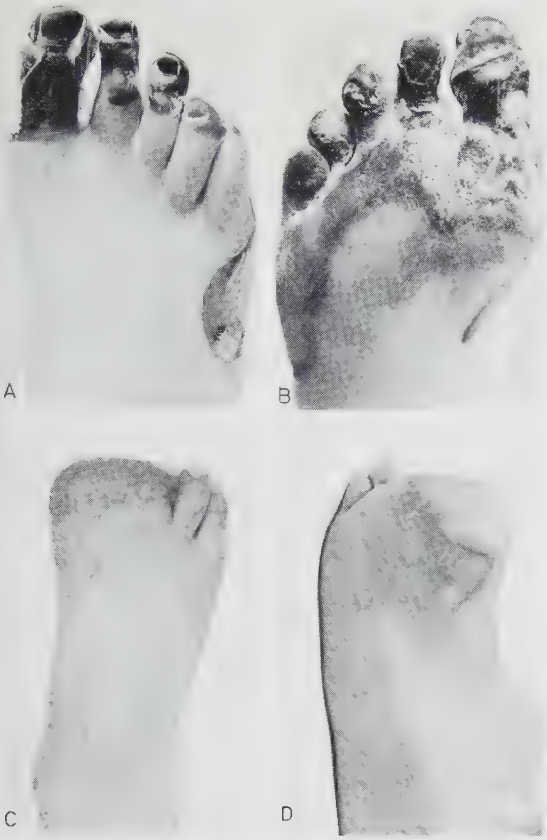



*Fig. 14.* Case 39. High tension burn in an engineer, aged 37. A current at 40,000 V passed from left arm to right foot. (A and B) Condition on admission 14 days after injury. (C and D) Final result 6 months later, after amputation of necrotic toes and cover by an abdominal tube pedicle. Note the normal appearance of the disto-lateral part of the sole and of the lateral toes, although these areas seemed to be of doubtful vitality as shown in pictures A and B.

*Fig. 15.* Case 46. High tension burn in an electrician. aged 53. A current at 70,000 V arced across to his left arm and earthed through the right lower leg. (A) 2 weeks after injury: Condition on transfer. (B) 4 weeks after injury: Condition after debridement showing extensive muscle loss with exposure of ankle joint. (C) 9 months after injury: Large abdominal tube pedicle being transferred to lower leg. (D) 2 years after injury: Pedicle inserted into lower leg. Irregularity due to partial loss of tube. Further trimming required.

had passed from one hand or arm to one of the lower extremities; bilateral lesions being caused by additional arc burns in 3 of the patients.

This group is characterized by severe lesions of the extremities, requiring prolonged hospitalization. Despite extensive surgery, crippling deformities resulted in a high degree of disability. In addition, 9 patients (cases 37, 40, 41, 43, 44, 45, 46, 47, 48) had peripheral nerve symptoms usually due to thermal injury.

Amputations were performed on 6 patients (cases 37, 38, 39, 43, 44, 48). Multiple amputations were carried out on one patient (case 43). Fig. 13. In this case, conservation of the right arm was considered possible as the circulation of the hand was relatively good, but arterial thrombosis resulted in gangrene of the hand 20 days after the accident.

In another patient (case 44) a right lower arm amputation was inevitable because of extensive deep tissue damage, although the circulation in the hand was quite good.

At operation 20 days after the accident the radial artery was found to be friable and was accidentally torn off at the level of the wrist. In the 4 other cases amputations were confined to fingers and toes.

In 5 patients (cases 39, 41, 42, 43, 48) deep lesions with exposure of tendons and bones, required primary repair with local or abdominal flaps and tube pedicles (Fig. 14). In 6 patients, split skin grafts were used for permanent or temporary repair. In 2 patients (cases 36, 40) no surgery was necessary.

Secondary repair, mainly consisting of scar excisions and split skin grafting, was performed on 9 patients. 3 of these, however (cases 43, 44, 46) were subjected to repeated and extensive surgical procedures. The former underwent 12 operations including nerve- and tendon grafting and tendon transfer. The other two underwent 11 and 18 operations respectively. The last case necessitated transfer of a large abdominal pedicle to the right lower leg where he had very severe burns with destruction of muscle and branches of the peroneal nerve (Fig. 15).

Capsulotomy of the m.p. joints of the right hand was performed in one patient (case 38), freeing of the median nerve in the right antecubital fossa in another (case 41). The latter had contracted renal failure and was treated with haemodialysis prior to admission to our Unit. His burns involved 15% of the body surface. Sequestrectomy of a partially amputated left thumb was performed in a third (case 48).

The average stay in the Burns Unit for primary repair was 60 days for 12 patients and the average healing time for all, including 2 outpatients (cases 36, 37) was 85 days.

Average incapacity for work for 10 cases, including one out-patient (case 37) was 490 days. One patient (case 48) is still undergoing treatment and has so far been unable to work for 8 months. In one patient (case 49), where secondary treatment had been delayed because of intercurrent disease, the incapacity for work was complete for 4 months and partial since that. One patient (case 45) was a boy, aged 10.

*Late results.* Nine patients (cases 37, 38, 39, 41, 42, 43, 44, 45, 46) have reported for examination and 3 (cases 36, 40, 47) have answered a questionnaire. Average observation time is 7 years (range 3 months–15 years). 7 patients (cases 36, 37, 39, 40, 42, 45, 47) stated that they were recovered, although they occasionally had some minor discomfort from scars.

Two patients (cases 37, 42) were judged clinically recovered, as no serious impairment of function could be detected. One patient (case 45) who was examined 2 years after the accident had some limitation of movement in the left shoulder joint, due to scars in the axilla, despite adequate primary skin grafting. He was therefore admitted for secondary correction. This patient also had a paresis of the left peroneal nerve, which will be discussed in a later chapter.

The mental processes of one patient (case 38) had been impaired since his accident 15 years previously. This had not been investigated, but the patient, aged 68 on follow up, refused any psychiatric consultation. He presented a picture of senile dementia and complained of loss of power in the right arm and limitation of movement in the index and middle fingers. The latter was mainly localized to the m. p. joints and was caused by deep fibrosis. There was marked loss of sensation and stereognosis in both palms, especially the right one. Split skin grafts on the thorax, arms and dorsum of both hands were of good quality, freely moveable, slightly hyperpigmented and with normal sensation. The disability was rated by the insurance company at 25%.

In one patient (case 39) the absence of the 3 medial toes caused a little trouble in walking. The abdominal pedicle graft to the distal part of the foot had poor sensation and was susceptible to pressure sores from ill fitting shoes. Along the junction between graft and plantar skin there was a tendency to fissure formation. Despite the loss of his toes, disability was rated at zero and he was able to continue in his work as an engineer.

Partial loss of function of the right median nerve persisted in one patient (case 41) 7 years after neurolysis was performed. He had atrophy of the biceps muscle and loss of stereognosis in the hand. A local flap in the antecubital fossa had excellent quality and normal sensation. A $8 \times 3$ cm split graft on the upper arm could hardly be distinguished from the surrounding skin. The sensation was poor however and the graft was adherent to the underlying muscle. The disability in this case was rated at 15%.

The patient with multiple amputations (case 43) had marked loss of sensation and movement in

the remaining thumb. There was no opponens function and he was unable to grasp. A volar pedicle graft on the remaining part of the hand was pigmented dark brown and devoid of sensation. This graft was very susceptible to minor injuries and fissures were observed along the junction with palmar skin. A split graft on the lower arm was partially adherent to the radius and was anaesthetic. Movement in elbow and shoulder joints was normal. This patient has to depend upon help from his family for the daily routine of dressing, eating etc. The insurance company rates the disability at 100%, but he is still able to play the trombone in a jazz band!

The other major amputee (case 44) had a stump partially covered with flap and split grafts of good quality and sensation. No pressure sores caused by the prosthesis were observed. A large split graft on the upper arm was adherent to the triceps muscle but caused no discomfort. Split grafts on the dorsum of the left hand could hardly be distinguished from the surrounding skin, and sensation was normal, as was the function of the hand. The patient was able to earn his living as an engineer. The disability was rated by the insurance company at 70%.

One patient (case 46) had a drop foot on the right side. This was partly caused by destruction of the extensor muscles and partly by a lesion of the peroneal nerve. The anterior aspect of the lower leg was adequately covered by an abdominal pedicle graft of good quality. Since the follow-up examination took place only 3 months after the last trimming of the graft, no sensation was observed. One year after the accident, Parkinsonism developed, although it is difficult to see a connection with the accident. This case will be discussed in a later chapter. The disability is not yet decided, and the patient has retired at the age of 55.

In 2 patients (cases 48, 49) still undergoing treatment, the disability is not decided, but in the former it is expected to be rated at 20% because of the loss of the left thumb.

*Comment.* Whether the current passes through both arms or one arm and one leg, the severity of the lesions seems to be the same. Lesions to the upper extremities however are naturally associated with a higher degree of disability, than those of the lower ones.

The high incidence of peripheral nerve symptoms in this group was mainly confined to the upper extremity. In most cases loss of nerve function was caused by thermal injury to the nerve trunk. These particular aspects will be discussed in a later chapter.

Observations on vascular damage in two cases should be mentioned:

In case 44, the radial artery was friable and brittle and as mentioned — torn off at the operation. At that time it could still maintain the blood supply of the hand, but it was probably only a question of time until spontaneous rupture would have taken place. Amputation was therefore made at a higher level where ligatures could be applied on healthy arteries.

In case 43 the right hand, although seriously burned, looked vital for almost 3 weeks when arterial thrombosis suddenly occurred and necessitated a lower arm amputation, despite anticoagulant thereapy being started as soon as the diagnosis was made.

The basic pathology is similar in these two types of complications, namely thermal damage to the vessel walls. In high tension burns this heat is generally so intense that widespread damage occurs despite the cooling effect of the circulating blood. These thermal effects are not confined to the area around the contact points, but vascular damage occurs deep to normal skin. Thrombi are apt to form within these damaged vessels. According to Muir (1958) ischaemic necrosis due to vascular thrombosis is common in high tension burns, and may dominate the clinical picture.

These cases illustrate two of the serious complications which one should be prepared for in high tension burns. Since one cannot accurately tell the depth and extent of an electrical burn in the acute stage, the prognosis should be guarded during the first 3 weeks.

*Pathway B*

This group (Table 10) comprises only 2 patients (cases 50, 51). In the former case the current entered through the left hand and earthed through both feet, and in the latter the current entered through both upper arms and earthed through the right foot and probably also the right gluteal region. Both patients were injured by currents at *relatively* low tensions and sustained small but deep burns.

*Fig 16.* Case 52. High tension burns in a boy, aged 16. The patient accidentally pushed an earthed metal ladder against a 16,000 V live wire, the current passed between hands and both feet. (A and B) Condition on admission

soon after injury. Note also the burn of the left wrist and the uninjured thenar and hypothenar emminences. (C and D) Condition 3 months after injury, following conservative treatment. Note the atrophy of thenar and hypothenar muscles, especially on the left side. Four years later sensation was normal in right hand and no atrophy was observed. In left hand atrophy still persisted and there was lack of intrinsic muscle function. Protective sensation present in left hand distal to the transverse scar in the palm. Loss of stereognosis. (E) Condition on admission soon after the injury. (F and G) 4 weeks later, following excision including amputation of the big toe and parts of the 1st metatarsal bone. (H and I) Final result 4 years later. The free skin grafts are adherent and lack sensation.

One of the patients (case 50) developed tetanus, the only case in our series. He was a farmer aged 45 who was admitted to our Unit a few hours after the accident. He was referred from the local hospital where tetanus toxoid had been given. 12 days later he developed tetanus which was treated successfully with serum and controlled ventilation combined with the administration of curare. Radical excision was performed, including amputation of the right 2nd toe. Skin grafting was carried out at a later stage and healing was complete 72 days after the accident.

The second patient (case 51) was referred from another hospital 34 days after the accident. The left triceps muscle was partially destroyed. Repeated split skin grafting only resulted in a partial take and the patient was discharged 46 days after admission, to await secondary healing. One year later scar excision and skin grafting of the left axilla was performed.

Average stay in the Burns Unit for primary repair was 59 days and the average healing time was 88 days. The average incapacity for work was 270 days.

*Late results.* The first patient (case 50) died from a cerebral hemorrhage due to malignant hypertension 12 years after the accident. $5\,^1/_2$ years after the accident he had been examined at our Unit, and at that time the result was judged excellent and there was no disability.

The other patient reported for examination after an interval of 9 years. Movement in the right shoulder joint was normal but motor power was poor owing to the loss of muscle. The split skin grafts on the left upper arm were thin, pale and smooth, without hypertrophic scars. The grafts were adherent to the remnants of the triceps muscle. Sensation in the grafts was poor, and certain areas when touched lightly, produced shooting pains into the lower arm. A split graft in the left sole was of very good quality, but the surrounding plantar skin had tender callosities. The disability was 33% as rated by the insurance company, and the patient had retired at the age of 58.

*Comment.* Since this group only comprises 2 patients it is hardly justifiable to make a comparison with the first group. However, the resistance of the current pathway in the present group is comparatively high and there is a definite tendency towards deep lesions associated with serious complications and disability.

Since the case of tetanus in our Unit in 1953, it has been our policy to give tetanus toxoid to all patients with burns, and in our opinion this is particularly important in electrical burns where tissue destruction is generally deep. Tetanus is rare in Sweden, probably because the majority of the population has been actively immunized.

*Pathway C*

This group (Table 10) also contains only 2 patients (cases 52, 53), but they illustrate how two accidents occuring under similar conditons may lead to quite different results. The patients accidentally touched a high tension wire with an earthed ladder and in both cases the current entered through the hands and earthed through the feet.

In the first case (Fig. 16 a) deep burns of both hands produced median and ulnar nerve damage but spontaneous healing took place. A burn of the left sole also healed spontaneously but the lesion on the right foot necessitated radical excision including amputation of the big toe along with $^3/_4$ of the first metatarsal bone. Fig. 16 b. Most of the soft tissues on the dorsum of the foot sloughed because of thrombosis of the arteria dorsalis pedis. The defect was temporarily covered with split skin. At a later date, the 2nd toe was amputated and further split grafts applied. During the period of treatment, function gradually returned to the right median and both ulnar nerves, but not to the left median nerve. The healing time was 129 days and primary treatment required a stay of 75 days in the Burns Unit. The incapacity for work was 485 days.

The other patient had full thickness burns of the left thenar region without damage to deeper structures. There were similar lesions on the dorsum of the right foot. A 3rd degree burn of the left big toe healed spontaneously, but deeper burns of the right big and 2nd toes required partial amputation. The burns of the thenar region and the dorsum of the right foot were covered by split skin grafts. In this case the healing time was 105 days and the hospitalization time for primary treatment was 38 days. The incapacity for work was 110 days.

*Late results.* Both patients reported for examination. The time since the accident was 3 years in the first case and 5 years in the second.

The first patient had complete anaesthesia in the median nerve distribution of the left palm, distal to the transverse scar. Sensation was good in the ulnar distribution. There was marked atrophy of the thenar and first dorsal interosseus muscles. Opponens function was poor. In the right palm there was a scar similar to that on the left hand, but sensation was almost normal and wrist and finger movement was good. In the left sole, a small scar caused no discomfort, but pro-

nounced hyperhidrosis was troublesome. The split grafts of the right lower leg and dorsum of the foot were adherent to the remnants of the extensor muscles and to the bones. These grafts were thin, dry, hyperpigmented and anaesthetic and consequently very susceptible to injury. The 3rd toe was markedly plantar flexed and slightly rotated medially. There was a pressure sore on the pulp. Correction of this deformity and flap repair of the foot was recommended, but the patient refused. The disability was rated at 40% by the insurance company.

The other patient was completely healed and had no complaints apart from occasional paresthesiae in the right foot. The graft in the left thumb web was slightly contracted but thumb movement was normal. Sensation was good in the graft. The graft on the right foot was thin, pale and smooth and a certain degree of hyperalgesia was observed. The shortening of the right big and 2nd toes was insignificant and he had no difficulty in walking. Disability rating was zero.

*Comment.* The first patient was using a metal ladder, and his hands and feet were wet with snow. The line voltage was 16,000 V. The other patient was using a wooden ladder but it was strengthened with iron and was well earthed by means of iron wheels. His hands were dry and he was wearing leather sandals. The line voltage was 70,000 V.

As the current always chooses the way of least resistance, we may assume that it preferably earthed through the ladder in both cases. It is however possible that the voltage drop in the wooden ladder was considerable, despite the iron bindings, thus exposing the patient to a much lower voltage than the rated one. The tension was sufficient to cause a break down of the comparatively high skin resistance at the contact points but not strong enough to produce deeper tissue destruction.

In the other case, although the voltage was only one quarter of that in case 53, contact was much better by virtue of the metal ladder and wet hands and feet. Consequently the lesions were much deeper and more extensive.

*Pathway D*

This group comprises 9 patients (Table 10), the majority of which were injured by currents at *relatively* low tension. In more than 50% the current traversed one arm or hand only. The burns encountered in this group were, as a whole, less severe and the healing time considerably shorter than in the other groups. Late sequelae were uncommon and no permanent disability was encountered.

Peripheral nerve symptoms occured twice, (cases 54, 56). In the former, partial loss of sensation in the left thumb was associated with small burns at the thumb base. Sensation returned in 12 days, by which time spontaneous healing had taken place. In the latter, transient paresis of both legs was reported immediately after the accident, but motor power returned after some hours.

This patient had a peculiar accident. He was working on a pole when the current (16,000 V) arced from the live wire to his left arm and earthed through his left side. He fell across the cantilever and lay in this position while the current was twice switched on by the automatic cut-out. The patient therefore received 3 high tension shocks before the current was finally turned off. On admission to our Unit 5 days later he had a large full thickness burn on the left side of the body, but surprisingly no damage to deeper structures.

An unusual complication occured in one patient (case 60) where the current had passed from the sternal region to the left arm. The burns presented no special problems but he was disorientated for one week after admission. EEG and ECG were normal. 23 days after the accident the patient suddenly became hoarse and on examination he was found to have paralysis of the left vocal cord. The patient had not shown any evidence of upper respiratory tract burns, nor had an endotracheal tube been used during the reconstructive procedures. Speech therapy was started and 6 weeks later the paralysis had recovered and the voice became normal.

Grafting operations were performed on 4 patients, split skin being used in 3 and in one an abdominal flap was transferred to the hand. Primary excision and closure was performed in one case and partial amputation of fingers in two others.

Secondary correction was required in two cases. Dense scar formation in the left axilla and left antecubital fossa in one patient (case 58) necessitated repeated grafting and intensive physiotherapy. Secondary repair entailed a stay of 87 days in our Unit.

The average stay in the Burns Unit for primary treatment was 29 days (8 patients). Average healing time for all patients was 54 days. Average incapacity for work was 79 days for 5 patients.

One was able to continue with his work. The rest of the patients were children.

*Late results.* Six patients have reported for examnation and one (case 54) has answered a questionnaire. One patient (case 56) could not be traced, and one patient (case 62) is excluded from follow up as he has only recently finished treatment. It should be mentioned that the latter is completely healed and no disability is expected.

Average observation time was 5 years (range 1–13). Five patients were completely recovered, including case 58 who had the axillary contraction. The scars however were still hypertrophic after the last operation, but movement was normal. The patient with the paralysis of the left recurrent nerve had not had any trouble with his throat since leaving hospital. Two boys (cases 57 and 59) had partially amputated index fingers, but otherwise had no disability. The patient that we were unable to trace at this follow up had been examined in our Unit 4 years after the accident and was then reported as being completely recovered.

*Comment.* As in low tension burns there was a striking difference in severity between injuries associated with current path A and those with current path D. However, the lower resistance in the latter group cannot be the entire explanation. A greater number of patients were injured by currents at lower tensions than in the former group. It is difficult to explain why the lesions in case 56 were not more severe after 3 shocks at 16,000 V. In this case the current path could not be accurately determined. There were no contact burns on his arm and it is believed that the current entered by arcing. Multiple electrical shocks are not uncommon and may occur under certain conditions in high tension installations, usually with disastrous results.

The recurrent laryngeal nerve palsy in case 60 is difficult to explain since intralaryngotracheal trauma does not appear to have occured. The entrance point of the current in the upper sternal region suggests a possible aetiology, but one would have expected the resulting injury to have manifested itself much earlier, whether thermal or neuronal in origin. There are two possibilities which cannot be ruled out. Arterial thrombosis and arterial rupture with haematoma formation, both due to damage to the vessel wall at the time of injury. A similar case was described by Audier & *al.* (1960); the current entered through the right orbit and earthed through both feet. These authors did not offer any satisfactory explanation for this peculiar lesion.

*Pathway E*

In the 3 cases constituting this group (Table 10) the current pathway could not be definitely ascertained, but it was probably from both hands to the back in 2 patients (cases 63, 64) and between the neck and legs in the third (case 65). In this group we had one fatal case and some unusual complications. Each case will therefore be described.

Case 63 was injured by current at 1700 V whilst standing on a lawn and sustained a small 3rd degree burn on the right scapular region. On admission to our Unit the same day he was complaining of upper back pain and X-ray revealed a compression fracture of Th6. This was treated conservatively and the patient was discharged on the 4th day. The burn healed spontaneously in 69 days, at which time he returned to work.

Case 64 was injured by current at 16,000 V whilst working on a pole. Strangely enough he did not fall, although both arms were completely paralyzed by the current shock. The paralyses gradually recovered over 3 weeks as also did the diminished sensation. On admission to our Unit 7 weeks after injury, granulating wounds and scars were found in the left elbow region. Excision and skin grafting was performed and the patient was discharged on the 15th day. Healing was complete 28 days later and he returned to work 160 days after the accident. The patient was troubled with tinnitus and loss of hearing from the date of injury and was referred for an ENT examination; audiogram confirmed deafness of neurogenic origin. He was treated repeatedly in the ENT department but showed no improvement.

Case 65 was also injured by a 16,000 V current whilst working on a pole. After the current shock he fell astride the lower cantilever. Five seconds after the first shock the automatic cut-out turned on the current again; the patient received further injuries and his clothes caught fire. The current was finally broken by earthing the live wire, and the patient was lowered to the ground after the flames had been extinguished. He was treated for 10 days in the local hospital and was then transported by helicopter to our Unit. He was found to have deep burns involving 60% of the body surface and was in poor conditon, disorientated and had pulmonary oedema. Despite intensive therapy the patient died from septicaemia (Staph. aureus and Pseudomonas) 5 days after admission.

*Late results.* The first patient (case 63) was completely recovered on follow up 7 years later. Case

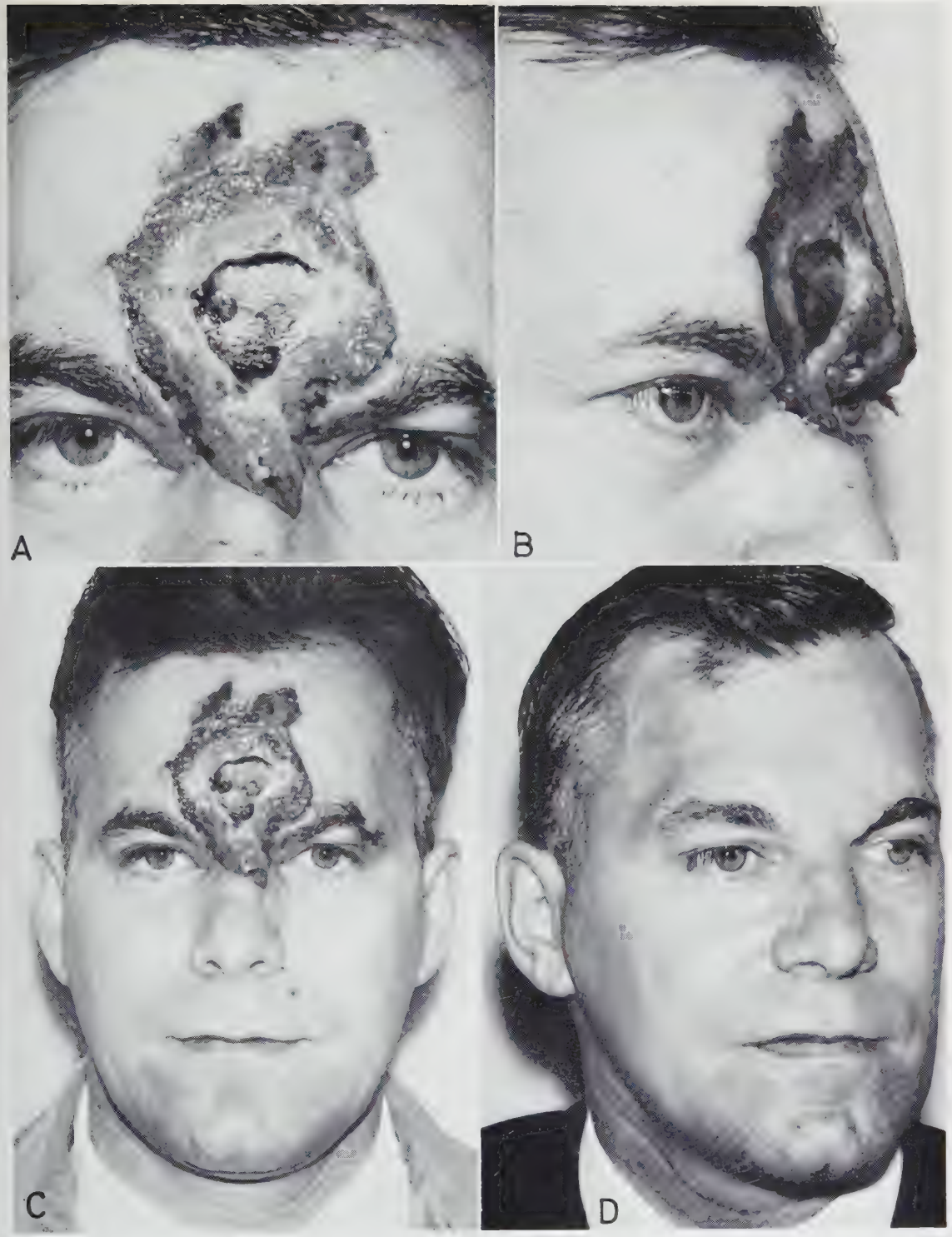



*Fig. 17.* Case 70. High tension burns (380 V) in an engineer, aged 42. (A, B and C) Condition on admission 6 weeks after injury (D) Final result 3 years later after removal of necrotic bone and repair with a forehead rotation flap. Secondary defect in the right temporal region is covered with a split skin graft.

64 had some loss of motor power in the right brachioradialis muscle. Split skin grafts around the left elbow were well matched to the surrounding skin and sensation was good. Tinnitus and deafness still caused discomfort and had been refractory to all treatment. These symptoms probably accounted for a disability rating of 15%.

*Comment.* The comparatively small local lesions in the first two cases can be explained by a current pathway of low resistance. The fatal outcome in case 65, was due to very unfortunate circumstances.

In our material the only fracture attributable to current shock was the spinal fracture in case 63. This was almost certainly caused by tetanic muscular contraction as a fall onto soft ground is unlikely to have been responsible. According to Kolar & Vrabec (1960) the mid-thoracic location of this fracture is typical of electrical accidents.

Tinnitus and deafness may occur after electrical accidents (Jemmi, 1965). Although a preexisting neurogenic deafness cannot be definitely excluded in case 64, the symptoms only became manifest for the first time shortly after the accident. According to Jemmi, derangement of the audito-vestibular apparatus can occur without direct current passage through the head. Pure vestibular symptoms tend to diminish owing to compensatory processes, but deafness shows a tendency to increase. The pathogenesis is obscure and there seems to be no effective therapy.

It is difficult to see how the fatal outcome in case 65 could have been prevented. The severe lesions produced by the two consecutive high tension shocks and the extensive arc and flame burns, were mostly 3rd degree.

*Pathway F*

This group comprises 11 patients who all had current pass through their head (Table 10). Strangely enough, the majority of burns involving the skull were not associated with cerebral complications, although there were some exceptions, 2 of them fatal. Sequelae and disability were in general, dictated by lesions other than the intracranial ones.

On account of the severity of the lesions, their prolonged healing time and the need for extensive surgical repair this group is comparable to the injuries associated with current pathway A. Some unusual complications and sequelae justify a more detailed description.

In 5 patients (cases 68, 69, 70, 72, 73) with burns of the scalp and skull, necrotic bone had to be chiseled away and the defect covered by a local flap (Fig. 17).

The duration of contact was apparently short with one exception (case 73; Fig. 12, Page 14), who lay unconscious, face down on the 380 V cables inside the control cabin of a crane, for about one minute. The current had passed from the left orbit to the right submandibular region. He was treated in the local hospital—where the left eye had to be removed—for 49 days and then transferred to our Unit. There was extensive, deep necrosis of the left orbital and temporal regions. As the necrotic bones were being chiseled off an epidural abscess was discovered and drained. The large defect was covered by acromiopectoral and temporal flaps. On the right side, resection of the angle of the mandible was later carried out because of osteitis which was causing painful trismus. This patient underwent 26 operations and stayed in hospital for a total of 400 days. Two years after the accident, symptoms of grand mal epilepsy appeared and EEG showed a focus in the left frontal lobe.

Case 75 was working on a pole when the high tension current arced across to his head and earthed through both feet. The patient fell several meters with his clothes on fire. On admission 4 hours later, he was restless and was complaining of pain in his head and right shoulder. He had deep burns on the anterior aspect of the trunk, face and lower extremities, involving 40% of the body surface. There were no clinical signs of intraabdominal injury, but microscopical haematuria was reported. Fluid therapy was instituted but after 48 hours he became oliguric and deteriorated rapidly despite intensive therapy. He died in anuria on the 5th day. Autopsy revealed contusion of the brain, two large ruptures of the liver, haemoperitoneum and haemothorax, casts in the renal tubules (probably myoglobin) and dislocation of the right sternoclavicular joint.

In case 76 the current pathway was the same as above. This patient sustained deep burns of the scalp, neck, trunk and extremities, involving 33% of the body surface. He was rendered unconscious and remained so for several hours. A tracheostomy was performed in the local hospital and he was transferred to our Unit on the same day. At no time after admission, was he able to maintain adequate oxygenation by his own efforts, and required continuous ventilation with the respirator. The patient remained in poor condition and repeated hemorrhages from the tracheostomy necessitated massive blood transfusions. The burns became heavily infected and a partial excision was performed as a last resort on the 16th day. There was a transient improvement, but he died two days later from cardiac arrest. At autopsy the most important findings were; deep coagulation of the brain and multiple erosions in the larynx and trachea.

*Fig. 18.* Case 72. Sequelae of high tension burn (6000 V) in an electrician, aged 21. (A) The condition on admission 4 months after injury, when the fingers were anaesthetic and immobile. Note pressure sores over the knuckles. On exploration the median and ulnar nerves were found to have been severed. After resecting back to healthy nerve tissue the gaps between their ends were 6 cm. Before suture both nerves were freed into the forearm and the ulnar nerve transposed at the elbow. The flexor tendons, which were stuck together at the wrist were freed. The area of nerve suture and tendolysis was covered by two local flaps, still visibile in B and C. (B and C) The result 3 years after injury. He can flex and extend his fingers fully, and there has been a reasonable return of protective sensation, but intrinsic muscle function is lost. Sublimis transfer for opposition of the thumb has been performed since the original exploration.

Other serious complications, although not endangering life, were observed in 3 patients (cases 66, 72, 74).

The first one, who had a small contact burn of the forehead, developed bilateral cataract 2 months after the injury. He was referred to the ophtalmology department and bilateral lens extraction was performed a few days later.

In case 72 the current passed from both hands to the head. He was treated for a total of 116 days in another hospital before admission to our Unit. At that time his right hand was "frozen" (Fig. 18). There was a typical claw deformity, with unstable scars around the wrist and pressure sores over the knuckles. There was total loss of sensation on the volar surface of the hand. A scalp burn involving the outer table of the skull presented no special therapeutic problems. The major nerve trunks at the right wrist had been completely destroyed. Resection and suture of the median and ulnar nerves was carried out, also secondary reconstructive procedures, including tendon transfer.

The third patient (case 74) whose facial lesions were more of the arc burn type showed a pronounced tendency to scar contraction despite adequate primary grafting (Fig. 19). Extensive loss of soft tissues in the nose and eyelids necessitated repeated grafting including acromiopectoral and forehead flaps. Despite this the scars remained unsightly and the relatively unprotected right eye developed keratitis. Multiple corrections were later required to correct the ectropion.

In 3 patients (cases 66, 67, 71) the scalp burns were insignificant and their treatment did not present any special problems. Case 71, however, also had a lesion of the spinal cord and will be discussed on page 48.

The average hospitalization time in the Burns Unit for primary treatment was 50 days for the surviving 9 patients in this group. The average total hospitalization time was 108 days. Average healing time in 8 patients was 110 days. In case 71 healing was not complete when he was transferred to a hospital near his home. Average incapacity for work was 486 days for the 8 patients.

*Late results.* Seven patients reported for examination, the average observation time being 4.3 years (range 2–11).

Four patients (cases 66, 68, 69, 70) were completetely recovered with excellent functional and cosmetic results. Split skin grafts on the skull, however, showed hyperpigmentation and had poor sensation. They were easily camouflaged by the hair style.

Case 72, with extensive nerve and tendon repair of his right hand had relatively good movement in the fingers, but intrinsic muscle function was lost. Protective sensation was present in the palm and fingers, but there was absence of stereognosis. His disability was rated by the insurance company at 33%. When treatment was completed, he returned to his work as an electrician for some years but he now runs a convalescent home which he finds more lucrative.

The patient who developed epilepsy (case 73) complained of constant headache but his grand mal attacks were kept under control by medication, and he only averaged two fits per year. The pedicle graft in the left orbital region matched the surrounding skin and sensation was good. The function of his mandibular joints was normal. A certain degree of mental impairment has complicated his rehabilitation and he can only take simple, part-time jobs. The disability rating is 66%.

Case 74 was very self-conscious about the ectropion and other defects on his face and has retired from all social events. On follow up, an EEG showed "unspecific abnormality" in the left temporal region. His compensation is based on a disability rating of 40%.

*Comment.* The relative resistance of the head is unknown when compared with that of the extremities, but judging by the local lesions it is probably about the same order. The multiplicity of symptoms, complications and sequelae encountered in this group, make it rather difficult to compare it with other groups. Serious cerebral complications occured in only two patients (cases 73 and 76). In the former, grand mal is probably not directly associated with the electrical injury, but rather with the epidural abscess. This is the only case of grand mal in our series. In the second case, sufficient heat was generated to produce cerebral coagulation. It is possible that the respiratory centre was also involved, hence the necessity for continuous ventilation on the respirator.

The brain contusion in case 75 is undoubtedly due to the fall, as were the other internal lesions.

Some rather mild mental complications were seen in our material but the incidence was not higher than is found in ordinary thermal burns (Körlof, 1965). Except in cases 73 and 74, there was no correlation between the current pathway and mental symptoms. Our observations are in keeping with those made by Bagozzi (1955–56) and Gatewood & Mc Carthy (1957). In accidents where the current passes through the head, thermal injury is mainly confined to the bones of the skull owing to their high resistance. The skull thus acts as a protective barrier for the meninges and brain.

The main cause of death in case 75 was a rapidly developing renal failure. This complication is seen more frequently than in thermal burns, and may occur when the total burned surface is not more than 10% (Moore, 1959). The deep tissue damage results in release of large amounts of myoglobin which block the tubules in the lower nephron. Myoglobin has been observed in the urine as early as 15 minutes after an electrical accident (Heidrich & Töppich, 1957). Alkali therapy has been suggested as a useful prophylactic against this type of renal failure, but the consensus of opinion is that adequate fluid therapy is more important (Gruber, 1963; Almgård *& al.* 1965). If renal failure occurs despite adequate fluid therapy, haemo—or peritoneal dialysis should be started. We did not have the facilities for haemodialysis, and peritoneal dialysis was out of the question on account of his abdominal burns.

*Arc burns*

Twenty-two patients had arc burns, mainly localized to the face and the hands (Table 11). The appearance of the burns, their clinical course and treatment did not differ from those of thermal burns. The majority of these patients had 2nd degree burns, involving less than 10% of the body surface. However, extensive 3rd degree burns were also encountered, which ended fatally in two cases. Six patients (cases 77–82) were treated as outpatients and 16 (cases 83–92) were admitted to the Burns Unit.

Skin grafting was required in three patients (cases 94, 95, 96) and in the latter there was deeper tissue damage involving the extensor tendons to the right middle and little fingers. The course in this case was also complicated by bilateral chondritis of the ears, resulting in partial loss of cartilage. This patient was later admitted for secondary correction, when tendon grafting to the fingers was performed.

*Fig. 19.* Case 74. Arc burns in a line worker, aged 41. (A) Condition 8 hours after injury. (B) 6 weeks after split skin grafting. Note the contraction and unpleasant appearance of the grafts of the right lower eyelid, cheek, nose and upper lip, and the destruction of the right eyebrow and nasal ala. (C and D) Final result, 3 years after the accident, following reconstruction of right side of upper lip and cheek with an acromio-pectoral tube pedicle. The nose and alar region have been reconstructed by means of a forehead flap. Eyebrow reconstructed using a narrow portion of the hairy scalp included in the upper margin of the forehead flap.

Fig. 19.

Table 11. *Patients referred for arc burns*

| Case number | Age years | Location of burns | Tot. surface of burns (%) | Sev. group | No opns. | Graft opns. | Tot. hosp. time (days) | Complications | Healing time (days) | Incapacity for work | Rated disability (%) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 77 | 41 | Hand | 1 | I | — | — | — | — | 14 | — | — |
| 78 | 26 | Hands | 1 | I | — | — | — | — | 14 | 21 | — |
| 79 | 46 | Face | 3 | I | — | — | — | — | 4 | 4 | — |
| 80 | 25 | Hands, face | 10 | I | — | — | — | — | 18 | 42 | — |
| 81 | 40 | Hand | 1 | I | — | — | — | — | 3 | — | — |
| 82 | 62 | Hands | 1 | I | — | — | — | — | 19 | 20 | — |
| 83 | 44 | Hands | 2 | I | — | — | 3 | — | 21 | 42 | — |
| 84 | 32 | Hands, face | 6 | I | — | — | 17 | — | 16 | 60 | — |
| 85 | 26 | Hand | 2 | I | — | — | 5 | — | 21 | 30 | — |
| 86 | 36 | Face | 3 | I | — | — | 2 | — | 10 | 10 | — |
| 87 | 30 | Face | 3 | I | — | — | 2 | — | 7 | 7 | — |
| 88 | 21 | Hand, face | 4 | I | — | — | 4 | — | 21 | 31 | — |
| 89 | 23 | Hands, face | 5 | I | — | — | 4 | — | 19 | 23 | — |
| 90 | 55 | Face | 5 | I | — | — | 3 | — | 17 | 21 | — |
| 91 | 64 | Face, neck | 9 | I | — | — | 5 | — | 68 | 70 | — |
| 92 | 53 | Hands, face | 10 | I | — | — | 19 | Nerve lesion | 35 | 227 | — |
| 93 | 44 | Hands, face | 10 | I | 1 | — | 22 | — | 73 | 214 | 10 |
| 94 | 22 | Legs | 18 | II | 4 | 1 | 43 | — | 99 | 128 | — |
| 95 | 19 | Hands, arms, face | 15 | II | 1 | 1 | 38 | — | 84 | 156 | — |
| 96 | 20 | Hands, arms, trunk, face | 30 | II | 7 | 2 | 100 | Loss of ext. tendons r. hand | 93 | 365 | 15 |
| 97 | 30 | Hands, arms, trunk, face | 38 | III | — | — | (4) | † | | | |
| 98 | 41 | Hands, arms, trunk, face | 50 | III | 1 | — | (6) | † | | | |
| Average | 36 | | | | 2.8 | 1.3 | 19 | | 33 | 82 | |

†: patient died.

Average hospitalization time for primary treatment was 15 days for the 14 surviving patients, and the average healing time for 20 patients was 32 days. Incapacity for work averaged 77 days for 19 patients. One outpatient (case 66) returned to work immediately after his first dressing.

Two patients (cases 97 and 98) were involved in the same accident. Arcing took place from a switch box causing extensive arc and flame burns in both patients. On admission 8 hours after injury the prognosis in case 97 was considered quite favourable as his burns were mostly superficial and not extensive. As he was in good general condition with normal blood values and adequate urinary output, fluid was given by mouth. Within a few hours he became hyper-pyrexial and oliguric and died in circulatory collapse on the following day. Autopsy revealed congestion of the lungs, liver and spleen, tubular nephritis and changes in the myocardium, possibly of ischaemic or toxic origin. No definite cause of death could be ascertained, but the pathologist felt that it was related to the myocardial changes.

The prognosis in case 98 was considered doubtful from the beginning as the burns involved 50% of the surface area and were mostly deep. Circumferential eschars of both arms prevented adequate blood supply to the hands and surgical decompression was performed 24 hours after admission with a temporary improvement in the circulation. The general condition remained poor and frequent haematemeses occured necessitating massive blood transfusion. The burns became infected with Staph. aureus and B. coli and the patient deteriorated rapidly, dying 6 days after admission. Autopsy showed sepsis and multiple erosions of the gastric mucosa.

*Late results.* Thirteen patients reported for examination and 3 answered a questionnaire. One patient who had died of carcinoma of the colon 2 years after the accident and 3 patients who had only recently finished treatment (but who are completely recovered) are excluded from follow up. Average observation time in 16 patients is 7 years (range 1–14). Eleven patients stated that they were completely recovered, and this was confirmed in 8 of them at follow up examination.

Case 92 had slight loss of sensation in the left hypothenar region, but otherwise had no sequelae.

Case 93 complained of loss of sensation around the mouth which caused discomfort in warm or cold weather. The dorsal skin of the hands was

Table 12. *Patients referred with sequelae of burns*

| Case number | Age on admission years | Tension (volt) | Current pathway | Location of sequelae | Type of sequelae | Incapacity for work (days) | Rated disability (%) |
|---|---|---|---|---|---|---|---|
| 99 | 8 | 220 | A | R. index finger | Scar contraction | 150 | — |
| 100 | 1 | 220 | A | L. 3rd finger | Klinodactylism | — | — |
| 101 | 7 | 220 | A | L. 3rd finger | Klinodactylism | — | — |
| 102 | 10 | 220 | A | R. & l. thumb web | Scar contraction | — | — |
| 103 | 9 | 220 | D | R. index finger & thumb web | Klinodactylism | — | — |
| 104 | 6 | 220 | D | R. index & 3rd finger | Scar contraction | — | — |
| 105 | 10 | 220 | D | L. index & 3rd finger | Cicatricial syndactylism | — | ? |
| 106 | 28 | 220 | D | L. index finger | Scar contraction | ? | — |
| 107 | 26 | 220 | ? | R. thumb web | Scar contraction | ? | ? |
| 108 | 7 | 220 | — | Lower lip | Scar | — | — |
| 109 | 6 | 220 | — | Lower lip | Scar | — | — |
| 110 | 17 | 220 | — | Lower lip | Scar | — | — |
| 111 | 16 | 220 | — | Lower lip | Scar | — | — |
| 112 | 15 | 220 | — | Lower lip | Scar | — | — |
| 113 | 9 | 220 | — | Mouth (r. commissure) | Scar | — | — |
| 114 | 14 | 220 | — | Lower lip | Scar | — | — |
| 115 | 4 | 220 | — | Upper lip | Scar | — | — |
| 116 | 17 | 220 | — | Lower lip | Scar | — | — |
| 117 | 14 | 220 | — | Lower lip | Scar | — | — |
| 118 | 57 | 3,460 | A | Both wrists | "Frozen hands" | 1205 | 75 |
| 119 | 24 | 11,500 | A | R. arm & r. lower leg | Loss of arm. Delayed healing | 248 | 65 |
| 120 | 54 | 20,000 | D | R. foot | Loss of big toe | 395 | 12 |
| 121 | 32 | 70,000 | D | R. foot | Loss of toe | 462 | — |
| 122 | 29 | 6,000 | E | R. shoulder & r. foot | Osteitis. Loss of toes | 1825 | 33 |
| Average | 17.5 | | | | | 714 | |

thin and dry following spontaneous healing of deep second degree burns. Because of this the insurance company had rated his disability at 10%.

In 3 patients (cases 94, 95, 96) skin grafts were slightly hyperpigmented, but smooth with almost normal sensation. In case 96 however, the skin on the dorsum of the hands was thin and unstable and there was some loss of motor power in both hands. His disability was rated at 15%.

*Comment.* As mentioned before, the burns in this group did not differ from other thermal burns; deep tissue damage only occurred once, causing loss of extensor tendons. The group as a whole did not present any of the special therapeutic problems encountered in contact burns.

There is no satisfactory explanation for the fatal outcome in case 97, although a pre-existing cardiomyopathy cannot be excluded. Renal failure supervened despite intensive fluid therapy and was unaffected by mannitol. It is difficult to decide whether the renal failure was primary and led to toxic damage of the myocardium or vice versa. Massive doses of antibiotics were given from the time of admission and there were no signs of sepsis at autopsy.

Sepsis was the main cause of death in case 98. A contributing cause may have been the gastric erosions.

## PATIENTS REFERRED FOR SECONDARY TREATMENT

Twenty-four patients were referred to us with sequelae from burns previously treated in other hospitals. Most of these burns were localized to the hands and lips, and in 80% of cases were caused by low tension (Table 12). The average interval between the accident to the commencement of secondary treatment in our Unit was 2.6 years (range $^1/_2$–17).

*Sequelae of low tension burns*

*Pathway A and D*

Four patients can be classified in current pathway A and another four in current pathway D. In one patient (case 107) the current pathway was unknown. As the complications and their treatment were essentially the same in both groups they will be considered together.

All these patients had contracted scars on one or more fingers resulting in varying deformities such as flexion contracture, klinodactylism, cicatricial syndactylism or contracted thumb web.

Excision or release of scars followed by full thickness skin grafting was performed on 7 cases, 2 of whom were treated as outpatients. In one case a pectoral flap was transferred to a thumb and in another a ring finger was amputated.

Average hospitalization time for 5 patients was 21 days. Surgery was not required in one patient.

*Late results.* Six patients reported for examination and 2 answered a questionnaire. One patient could not be traced. Average observation time was 6.3 years (range 1–14).

All patients followed up were functionally improved, Slight limitation of movement in one or more fingers caused no disability. The grafts were all of good quality, with almost normal sensation, but slightly hyperpigmented.

*Special Pathway*

Ten patients, mostly girls in their teens were referred with scars on the lips resulting from burns sustained in early childhood. In one case the scar was localized to the right commissure (Fig. 20), in one to the upper lip, and in the other cases to the lower one.

In 8 patients (109–116) scar excision was carried out and the defect repaired by a local flap. In one patient (case 117) a median scar of the lower lip was excised and the defect covered with a postauricular skin graft. The average hospitalization time for 6 patients was 7 days. Two were treated as outpatients and in one case no surgery was necessary.

Additional corrections were required in 2 patients (cases 112, 115) the latter having been referred to another plastic surgery unit when he moved to a different county.

*Late results.* Five patients reported for examination and 3 answered a questionnaire. Records were sent from the hospital which took over the treatment in case 115. One patient could not be traced. Average observation time was 7.3 years (range 1–12).

In the five patients examined, the cosmetic result was good. The scars were barely visible when the mouth was in repose but pouting and attempts at whistling disclosed a small muscular defect in two patients. The questionnaire patients stated that they were not satisfied with the appearance of the lip, although they did not volunteer further information. Reports on the patient who was treated at the other hospital state that he has required 2 minor corrections to the Cupid's bow, the last one being 1 year ago.

*Comment.* Secondary deformities of the fingers after deep burns were partly due to scar contraction, but aggravated in growing fingers by disturbance of growth. This was particularly noticeable in some of the younger patients with long-standing sequelae. It seems important therefore not to delay the release of scar bands or the provision of adequate skin cover if these deformities are to be avoided.

The preponderance of females referred with lip complications does not necessarily imply that these injuries are more common in girls, but is probably due to the greater vanity of female teenagers. Larger series have actually shown a preponderance of boys (Thomson & *al.*, 1965; Fogh-Andersen & Sørensen, 1966). The usual location on the lower lip is possibly explained by the fact that the "short" takes place before the child can close his mouth. It is surprising how large tissue losses in the lip can heal spontaneously, causing *relatively* little deformity. This fact supports the view that extensive repairs should not be undertaken during the acute stage but should be delayed until healing is complete and the scar tissues have softened (Schuchardt, 1958; Kazanjian & Converse, 1959; Fogh-Andersen & Sørensen, 1966 and others). It is noteworthy that no disturbance of speech, dentition or development of the lower jaw was observed in the patients followed up.

*Sequelae of high tension burns*

Five patients were referred with sequelae of high tension burns localized to the extremities. The average time between the accident and admission to the Burns Unit was 2.6 years (range $^{3}/_{4}$–8).

*Fig. 20.* Case 113. Low tension burn in a girl, aged 1. The patient sucked the free end of a live extension cord. (A) Condition several weeks after injury when the patient was treated in another hospital. (B) Condition on admission for secondary repair 9 years after injury. Large hypertrophic scar following spontaneous healing. Loss of tissue seems small despite the large primary defect. (C and D) 18 years after injury. Scar excision, suture and commissurotomy have been performed. Photos taken one year after the last operation.

*Pathway A*

Two patients were admitted (cases 118, 119). The former had suffered identical lesions on both wrists when, 9 months earlier, he had been traversed by current at 6000 V. He presented a most crippling condition with two "frozen" and anaes-

thetic hands. He was unable to feed and dress himself. The fibrotic ends of the severed median and ulnar nerves were resected and continuity restored by suture, thereby achieving a return of protective sensation. The function of the hands was partly restored by additional operations, including extensive tenolysis of the profundus tendons with resection of the sublimis, tendon transfers to the thumbs, arthrodesis and arthroplasty of the p.i.p. joints and capsulotomies of the m.p. joints. This patient spent a total of 77 days in hospital and underwent 15 operations.

In the second patient, the current had passed from the right arm to the left leg, resulting in extensive and deep burns which necessitated an upper arm amputation 20 days later. When first seen 1 1/2 years later he had a large granulating ulcer in the region of the tibial tubercle. The wound was excised and covered with a cross leg flap.

*Pathway C*

Two patients (cases 120, 121) in whom the current had passed from both hands to both feet, were admitted 8 and 1 year respectively after the accident. Burns were localized to the feet, but in neither case were they severe and treatment was limited to amputation of the big toe in the former and the second toe in the latter.

*Pathway E*

One patient (case 122) was traversed by current at 6000 V from the right scapular region to both feet. When admitted to our Unit almost 2 years later he had necrotic areas involving muscle and bone over the right shoulder, thin unstable scars on the dorsum of the right foot and loss of the little toe. After excising the scar and dead bone, the defect was covered with a rotation flap from the lower scapular region. Scar tissue was excised from the anterior aspect of the lower leg and dorsum of the foot and cover was obtained by a crossleg flap and additional free grafts. Subsequent tendon grafting was required to correct the drop-foot deformity. Minor corrections, including amputation of the 3rd toe were also performed. He stayed for a total of 123 days in hospital.

*Late results.* All 5 patients reported for examination, the observation time being on average 7 years (range 4–11).

Case 118 still had difficulties with feeding and dressing but he was able to continue his work as a supervisor in a power station. Movement was markedly restricted in all fingers and intrinsic muscle function was lost. There was protective sensation in both palms but no stereognosis. Disability was rated at 75%.

Case 119 was severely handicapped in his daily routine but was able to earn his living as an engineer. The amputation stump was sound and movement in the shoulder joint was normal. Motor power was reduced in the right leg. The grafts below the right knee had poor sensation and were susceptible to injury. Disability was rated at 65%.

Case 121 had only minor discomfort from an ankylosed big toe and was in full-time occupation as a line worker.

Case 120 occasionally complained of aching in the right leg probably as a result of thrombophlebitis a few years previously. He also complained of weakness in the right arm. On examination, motor power in the right hand was only half of that in the left one, but sensation was normal. There was pronounced hyperkeratosis at the amputation site of the big toe, but motor power and sensation were good in both feet. The patient was unable to climb ladders and had therefore given up his job, but was re-employed as a research engineer. The disability was rated at 12%.

Case 122 complained of pain in the right foot where the graft was adherent to bone. It had poor sensation and it was necessary for him to wear a protective dressing to prevent ulceration. Movement in the ankle joint was markedly restricted, with a tendency to drop foot. However, the gait was almost normal. He had been able to continue his work as an electrician but was always given light work. The flap over the right scapular region had excellent appearance with normal sensation. The free grafts on the secondary defect had the same colour as the surrounding skin and were smooth, but with somewhat impaired sensation. Movement in the right shoulder joint was slightly restricted on account of webbing in the posterior axillary fold, but correction was considered unnecessary as the patient had no discomfort. Disability rated at 33%.

*Comment.* In these 5 cases with sequelae of high tension burns two things are striking. Firstly; le-

sions associated with current pathway A were more extensive and disabling than those associated with current pathway C where the resistance is lower. This confirms the findings in patients referred for primary treatment; namely, the greater the resistance, the more severe the local lesion.

Secondly; the time between the accident and referral to the Burns Unit was unnecessarily long in some cases. It has been shown (Skoog, 1963) that full thickness burns admitted for primary treatment had an average stay in our Unit of 51 days whether they were admitted within 48 hours or referred after 180 days. These figures indicate that full thickness burns require treatment in a specialized unit and this is even more essential in electrical burns.

## TREATMENT

### *General care of the patient*

In our Unit the general treatment of electrically burned patients follows the same principles used in thermal burns. Only a few points with special relevance to electrical burns will be stressed.

Special attention is paid to the fluid balance, as electrical burns tend to go into renal failure more readily than thermal burns of equal severity.

Because electrical burns tend to be deep we consider tetanus prophylaxis of paramount importance and toxoid is given routinely.

ECG and EEG examinations are performed provided they are not contraindicated by the type and location of the burns.

### *Local treatment of the burn wound*

Arc burns are treated as other thermal burns, but the local treatment of contact burns is different. Some authors (Jellinek, 1936, 1960, and Dale 1954), recommend extreme conservatism; waiting for spontaneous separation of the eschar, before skin grafting. The rationale for this is apparently the poor result often obtained by primary excision and skin grafting, first introduced by Wells in 1929. Muir (1958) feels that the progressive necrosis generally responsible for these failures is not specific to the action of electricity, but is due to secondary bacterial invasion of tissues partially devitalized by the current. A distinction should be made between this type of necrosis and that due to arterial thrombosis which produces death of tissue outside the current pathway. The present trend seems to be a modified early surgical treatment (Artz & Reiss, 1957; Michon & Vichard, 1962; Hjern *& al.* 1966).

All our burns are treated by the exposure method. This has proven particularly useful in electrical burns which usually become dry during the first week. This facilitates physiotherapy which is instituted on the first day and continued throughout treatment, with the exception of the immediate post-graft period. The importance of physiotherapy in these cases cannot be emphasized too strongly; contractures develop rapidly and once established, are difficult to release. Lesions of the wrist are especially liable to contractures. First excision has been performed on average 15 days after the accident. However we do not adhere to a rigid schedule, as the optimum timing of excision and grafting depends upon the extent and localization of tissue damage. In deep burns, repeated excisions have to be performed to avoid removal of any viable tissue. Wet dressings with 0.5 per cent aqueous solution of aluminium subacetate accelerate separation of the eschar, and during the sloughing stage daily baths are often given. Burns not extending deeper than the subcutaneous layer are generally ready for grafting in about 3 weeks. The average time between the accident and first grafting was 25 days in this series. The time spent on repeated conservative excisions is worthwhile when the ultimate results are evaluated. Whenever possible split skin is preferred in the first instance, either permanently or as temporary cover. If tendons, nerves or bones are exposed, flaps are used, preferably from a distant site where tissue damage due to the current is unlikely to have occurred. A flap closes the wound and at the same time counteracts progressive necrosis. If local flaps are available we do not hesitate to excise and provide cover at an early stage. In burns of the hand we have been more inclined towards a conservative approach, although the excellent results reported by Peterson (1966) show that early surgical treatment has many advantages.

Where circumferential burns of the extremities endanger peripheral blood supply, surgical decompression is performed by one or two longitudinal incisions through the eschar. This should be done within the first 24 hours as delay may result in loss of the extremity, and toxins under the eschar

may build up to dangerous levels before they are released into the general circulation (Skoog, 1963).

Primary excision and simple closure have occasionally been carried out in small, low tension burns, with good results. This should however not be done in high tension burns, where progressive necrosis is liable to occur.

Adequate treatment of electrical burns usually entails much more than providing skin cover. To achieve a good functional result reconstructive procedures upon nerves, tendons, bones and joints were often required in our patients. Amputation may also be necessary, as a lifesaving procedure. Treatment that is too conservative can result in serious infection and toxemia.

The hospitalization time of our electrically burned patients has been reduced in recent years. This is partly because excision and grafting are now carried out earlier than before. We have also found it advantageous to discharge the patient after primary healing and then readmit him for reconstructive surgery.

## DISCUSSION AND CONCLUSIONS

When current flows through a conductor of homogenous material and uniform cross section, the voltage drop due to resistance is directly related to the length of the conductor. A voltage drop also occurs when current flows through the human body but it is not uniform along the current pathway owing to variations in tissue resistance and cross sectional area.

According to Freiberger (1934) and Osypka (1963) the internal body resistance is largely determined by the limbs on account of their content of bone, joint and tendon. The relative amount of these three structures — and consequently the resistance—increases in the distal part of the limb. This is of great practical importance as hands and feet are most often the parts of the body which complete the circuit between two contacts, or between one contact and earth. This combination of high internal resistance and comparatively small cross section causes a considerable voltage drop. The transformation of electrical energy into heat is therefore maximal in the skin at the contact site and in the adjacent tissues. As the tissue resistance of the trunk is less and its cross sectional area much greater, the amount of heat released is considerably smaller and thermal damage to internal organs is very rare.

As mentioned above the voltage drop and the resultant release of heat along a pathway of varying resistance is directly proportional to the latter at any one point. This fact prompted a study of the severity of burns and internal damage in relation to current pathways of different resistance. Any classification based upon current pathways, must of necessity be rather indefinite, as many factors such as voltage, duration of current flow, concomitant trauma — and perhaps also individual susceptibility — must be taken into account. Even if this classification of clinical material hardly stands comparison with the cadaver experiments carried out under good, standardized conditions by Freiberger and Osypka, it is based upon fundamental principles of physics.

According to the circuit diagram in fig. 3, p. 8 the most severe local lesions would be expected with current flow through the pathway of greatest resistance, i.e. pathway A (Hand→hand, or hand →foot) while current flow through the low resistance pathway D (in—out the same extremity) would be expected to cause less serious damage. The two major groups of low tension burns were associated with current pathways A and D. In the former the resistance is about four times that of the latter and a striking difference in depth of the lesions and in the clinical course was observed between the two groups. Burns associated with current pathway A were more severe, had a higher incidence of complications and more often required hospitalization and surgical treatment.

The same pattern was discernable in high tension burns, although additional arc and flame burns sometimes made accurate analysis difficult. On account of the relatively small number of patients injured via current pathways B, C and E a comparison is hardly justifiable, but a similar tendency was noticed; the higher the resistance of the current pathway, the more severe the burns tended to be and the greater the disability. It should be mentioned that local lesions were similar whether the current passed between both hands or between one hand and one foot. However, burns of the hands naturally accounted for a greater degree of disability than those of the feet.

The nature of scalp and skull burns suggests a high resistance at the contact site. The resist-

ance of the cranial bones is probably due to the outer and inner table, as the highly vascular diploe is supposed to be a good conductor. The large voltage drop in the skin, galea and outer table results in the heat being largely dissipated outside the brain, thus sparing this vital organ from direct thermal injury. This probably explains the surprising fact that these burns were not often associated with serious cerebral complications. Despite this voltage drop in the external layers of the skull however, the amount of electricity passing through the brain was still sufficient to produce unconsciousness in every case. The moderate degree of mental disturbance in most cases was not necessarily considered evidence of intracranial electrical damage, as the symptoms were essentially similar to those observed in patients with purely thermal burns.

Although the rate of current flow through the trunk is generally insufficient to cause thermal injury to internal organs, the latter may suffer serious functional derangement. The heart is of course the most susceptible and a fatal disturbance of the pacemaker mechanism may occur with current values in the region of 1/100 to 1/200 Coulomb (Von Holstein-Rathlou, 1936). This is equivalent to 50 or 100 mA for a fraction of a second. Sudden death may result from ventricular fibrillation. If the patient survives the electrical shock, transient disturbances of the conduction system may sometimes occur.

In our series there were no cases of physiological disturbances in internal organs (with 2 exceptions in which the symptoms were mild and transient). This is surprising, as one would expect a high amperage through the trunk on account of its low resistance. It may well be that the margin between a current which produces relatively unimportant cardiac irregularities and one which causes fatal ventricular fibrillation, is very small.

The infrequency of electrical cataract was surprising in view of the fact that deep contact burns of the forehead were fairly common. This complication occurred only once and was associated with a relatively superficial burn. It must be borne in mind that cataract can occur many years after the accident, but in none of our other patients in this group—reviewed for periods up to 15 years—was there any evidence of lens opacity.

Other general and local complications encountered in this material were as a rule independent of the current pathway.

The mid-thoracic spinal fracture in one case occured when the current passed from both hands to the back. This typical complication is liable to occur with any pathway through the trunk. It may also occur during convulsions induced by current flow through the brain, similar to those produced by electroshock therapy.

Arterial damage—leading to either haemorrhage or thrombosis—occured irrespective of site. Although the damage was mainly thermal, secondary infection very often played a part, particularly where the vessel lay superficially. We have seen repeatedly that high tension currents tend to pass through soft tissues without being deflected by anatomical structures. It is possible however, that low tension currents may tend to follow blood vessels although it was not possible to confirm this view in our clinical material.

Electrical injury to a blood vessel does not necessarily imply that it is a better conductor than the adjacent tissues, but rather that its specialized function is destroyed by trauma which would be insignificant in a tendon or muscle. This also applied, in greater degree to peripheral nerve injuries, and will be discussed in the next chapter, in more detail.

One frequently encounters in the literature (North, 1948; Baldridge, 1954; Lambusta & Catalano, 1961) the statement that electrical burns seldom, if ever, become infected. This has possibly originated in a statement by Jellinek (1955, 1962) to the affect that the electrical *wound* never becomes infected, although the *burn* does. He made a plea for a specific electromechanical, local injury which can occur independently of burns. He showed some of these lesions in his papers. They do not seem to have the characteristics of burns, and are said to heal without infection. Lesions of this type did not occur in our material and in our opinion electrical burns are as prone to infection as thermal burns, but possibly at a later stage. Infections which are reputed to be common in electrical burns are tetanus and gas gangrene. We had no case of gas gangrene in our series and only one of tetanus. The latter being the only case ever to occur in our Burns Unit.

For many years the local treatment of electrical injuries has been hindered by prejudices and mys-

ticism, and to many doctors the electrical burn has been a noli me tangere. Conservative treatment may be an advantage in selected cases but the experience gained from this material justifies the conclusion that whenever possible treatment should be carried out early, according to the principles of reconstructive surgery.

Electrical trauma is a complex lesion, comprising a general injury to the whole organism with symptoms from various organs, plus a special type of burn. The prognostic and therapeutic problems are manifold and in handling these patients, cooperation with other specialists is sometimes essential. Electrical burns should therefore be treated in a Burns Centre. This will not only provide a better service but will also broaden the knowledge concerning these uncommon but important injuries.

An analysis of the aetiology of the accidents in our series shows that most of them could have been prevented. This applies particularly to children with low tension burns acquired at home. Defective plugs and sockets and inadequately insulated cables were, by far the commonest cause of these accidents. In recent years following the introduction of stringent regulations and better insulating material we have observed a marked decrease in the accident rate. In many homes, however, electric appliancies of the old type are still in use and constitute a constant danger. Such equipment should be replaced by cables with plastic insulation and by non-detachable plugs and un-breakable sockets. Detailed information about electrical hazards should be brought to people through television, radio and press.

The high tension injuries affected mainly electricians and other technically skilled persons who were quite aware of the hazards. Many of these accidents could also have been avoided. Lack of attention, fatigue, carelessness and misunderstanding were responsible for more accidents than technical failures.

High tension injuries in young boys are very difficult to prevent. Boys always take a delight in trespassing and although they may be able to read a warning notice, it does not convey any idea of the danger. In railway stations and shunting yards the lines must of necessity be high voltage and it is difficult for the personnel to keep a continuous strict watch over such areas. Educational talks in school at an early age are perhaps the only way to reduce the number of these accidents.

# PERIPHERAL NERVE INJURIES IN ELECTRICAL BURNS

S. Blom and O. M. Ugland

A survey of the available literature on electrical injuries of peripheral nerves reveals that most papers are solitary case reports of many years standing based only upon clinical observations. The extent of peripheral nerve injury is therefore difficult to evaluate. Panse (1955), gave a critical review of the literature on the subject up to that time. In his large monograph on the neurology of electrical trauma he deals mainly with the effects on the central nervous system. However, he expressed the view that permanent damage to peripheral nerves can always be traced to thermal effects. Transient loss of nerve function is, according to Panse, related to rapidly reversible changes in the spinal cord. No experimental or other proof of these statements has been produced. More recent literature (Panse & Menden, 1962; Courville, 1963; Silversides, 1964) has not thrown any more light on these problems.

It is certain that transient impairment of function may be attributed to direct action anywhere along the course of the peripheral neuron by the current itself. Furthermore, permanent loss of nerve function could be the result of damage to the blood vessels supplying the nerve, as well as scar formation in surrounding tissues with secondary involvement of nerve fibres.

The aim of the investigation reported here was to study the following problems:

1. How common is peripheral nerve dysfunction in acute and recent cases of electrical injuries?
2. To what extent will electrical lesions cause permanent damage to the peripheral nerves?
3. Can any conclusions be reached with the method used regarding the pathogenesis of neural dysfunction following electrical injuries?

## MATERIAL

*Group I.* Eight males (Table 13) examined during the period of primary treatment constitute this "acute" group. The time between the accident and the first examination varied from 2 hours to 21 days. The average age of the patients was 29.2 years (range 2–58).

*Group. II.* Twenty-four patients injured more than one year earlier were selected for neurological and neurophysiological follow up (Tables

Table 13. *Patients subjected to clinical neurological and neurophysiological examination during the period of primary treatment. Group I*

| Case number | Age years | Tension (Volt) | Current pathway | Location of burns | Immediate symptoms |
|---|---|---|---|---|---|
| 4 | 2 | 220 | A | Hand | None |
| 45 | 10 | 16,000 | A | Head, neck, u. extr., trunk | Unconsciousness |
| 46 | 53 | 70,000 | A | Arm, trunk, leg | Paresis r. leg |
| 47 | 40 | 6,000 | A | Hand, arm, leg | Paresis r. arm. Unconsciousness |
| 48 | 36 | 500 | A | Hands, trunk | Unconsciousness |
| 49 | 24 | 16,000 | A | Hands, arm, trunk | Unconsciousness |
| 61 | 58 | 6,000 | D | Arm, trunk | None |
| 71 | 11 | 16,000 | F | Head, neck, trunk, legs | Unconsciousness |

Table 14. *Follow up cases without initial abnormal neurological symptoms. Group IIa*

| Case number | Age on follow up years | Tension (Volt) | Current Pathway | Location of burns | Sequelae of burns |
|---|---|---|---|---|---|
| 1 | 21 | 220 | A | Hands | None |
| 3 | 7 | 220 | A | Hands | None |
| 5 | 10 | 220 | A | Hand | Amp. r. 5th finger |
| 6 | 16 | 220 | A | Hand | Amp. r. 4th finger |
| 10 | 39 | 220 | A | Hand | None |
| 11 | 7 | 220 | A | Hands | None |
| 23 | 5 | 220 | D | Hand | None |
| 59 | 4 | 220 | D | Hand | Klinodactylism index finger |
| 92 | 61 | 7,500 | Arcing | Head, hands | Impaired sensation l. hand |
| 93 | 49 | 380 | Arcing | Head, arms, hands | Loss of sensation around the mouth |
| 94 | 27 | 380 | Arcing | Legs | None |
| 96 | 26 | 380 | Arcing | Head, trunk, u. & l. extr. | Loss of extensor function r. 5th finger |

14, 15). Two patients were females, aged 7 and 16 years respectively. The average age of the patients was 31 years (range 4–61) and the average observation time was 5.7 years (range 1–14). This is a heterogenous group subjected to voltages varying from 220 to 70,000. Some patients were traversed by the current, others were exposed to arcing only. Some had initial neurological symptoms (unconsciousness, paresthesiae, pareses, etc.). The group includes patients with late sequelae as well as those who were completely healed. Patients who had operations on main nerve trunks were excluded. For practical reasons it was not possible to fully investigate the entire series and attempts were made to achieve a sample representation of this heterogenous material.

Table 15. *Follow up cases with initial abnormal neurological symptoms. Group IIb*

| Case number | Age on follow up years | Tension (Volt) | Current Pathway | Location of burns | Immediate symptoms | Sequelae of burns |
|---|---|---|---|---|---|---|
| 27 | 60 | 220 | D | Hand | Unconsciousness | None |
| 29 | 16 | 220 | D | Hand | Loss of sens. r. thumb | None |
| 39 | 45 | 40,000 | A | Hand, arm, foot | Unconsciousness, convulsions | Amp. 1st, 2nd, 3rd, toes r. foot |
| 44 | 43 | 10,500 | A | Head, neck, hand, arm, trunk, leg | Unconsciousness, paresis r. arm | Amp. r. lower arm |
| 52 | 20 | 16,000 | C | Hands, feet | Unconsciousness, pareses both hands | Loss of function r. median. nerve. Amp. 1st, 2nd toe. |
| 53 | 34 | 70,000 | C | Hand, arm, trunk, feet | Unconsciousness | None |
| 64 | 48 | 16,000 | E | Hands, arms, trunk | Paresis both arms | Weakness r. arm, mental troubles |
| 66 | 58 | 6,000 | F | Head, trunk, arms, hands | Unconsciousness, agitation | Cataract |
| 69 | 34 | 70,000 | F | Head, hand, arm, trunk, feet | Unconsciousness, agitation, paresis l. leg | Amp. both big toes |
| 74 | 51 | 20,000 | F | Head, trunk, arm, hand | Unconsciousness | Scar contractions, depression |
| 119 | 29 | 11,500 | A | Hand, arm, leg | Unconsciousness, paresis r. arm | Amp. r. upper arm |
| 122 | 40 | 6,000 | E | Trunk, feet | Paresis both feet | Amp. r. 1st, 3rd, 5th toes |

## METHODS

*1. Clinical examination*

A clinical neurological examination was performed in most cases. The patients were questioned about loss of sensation and muscle weakness. Atrophy in different muscle groups was sought and reflexes were tested. Motor power was tested in the extremities and in some cases in the facial muscles also. Cutaneous sensation was tested.

*2. Electromyographic examination*

Concentric needle electrodes (Disa 13 K 0031) were used. Special attention was paid to the EMG examination in those muscles where denervation was suspected, either because of the nature of the original trauma or because of the clinical findings. The activity was displayed on an oscilloscope and studied in the ordinary way during relaxation, and moderate and maximal voluntary contraction. EMG was not performed on patients who had no initial neurological symptoms or signs or on those with normal neurological findings at follow up. EMG was not performed routinely on emergency cases, partly because of its limited value in the early stage of nerve injury, but also because the general condition of the patient and the location of their burns very often prevented an extensive examination with needle electrodes.

*3. Motor conduction velocity study*

Surface electrodes were used. A soaked pad electrode with a diameter of 15 mm served as cathode. It was placed over the nerve to be stimulated, a large anode being fixed to the opposite side of the extremity under investigation. The bipolar surface recording electrode had an interelectrode distance of 23 mm (Disa 13 K 62) and was placed over the thenar and hypothenar muscles or the short toe extensor muscle, depending on the nerve to be examined. A ground electrode was placed over the wrist or ankle joint as close to the recording electrode as possible.

The stimuli were rectangular pulses with a duration of 0.5 msec. delivered at intervals of about 2 seconds, 4 or 5 stimuli being applied at each point of stimulation. The stimulus strength was adjusted until no further increase in amplitude of the response occured. The evoked muscle potential was displayed on an oscilloscope and photographed. The conduction velocity of the fastest conducting fibres of the nerve under study was calculated in the usual manner. The distance between the proximal and distal point of stimulation was measured as accurately as possible.

The intramuscular temperature was not measured. The temperature of the room was usually about 25°C.

Under the experimental conditions described, the lower limits of normal conduction velocity were accepted as being 45 m/sec in the median and ulnar nerves and 40 m/sec in the peroneal nerve.

## RESULTS

*Group I*

In 3 patients (cases 4, 48, 61) who were examined 1, 7 and 21 days after the accident respectively, there were no abnormal neurological signs and conduction velocities in the involved extremities were normal. In the last case EMG was also normal.

Various signs observed in the other patients in this group will be described in more detail.

*Case 45.* This boy aged 10, threw a metal wire over the 16,000 V live cable whilst standing on the roof of a coach at a railway station. The current passed through the upper part of the body between both hands causing extensive burns of the trunk and upper extremities, and the patient fell unconscious to the ground. On admission 5 hours later, no abnormal neurological signs were observed, apart from limitation of movement in hands and arms due to pain. A haematoma of the left trochanteric region was observed but motor power and sensation were normal in the lower extremities. Seven days after the accident, conduction velocities were determined. It proved impossible to excite the left peroneal nerve despite good voluntary action of the anterior tibial muscle and the short toe extensor. Conduction velocities were normal in the right peroneal and both ulnar nerves. EMG performed 17 days after the accident, revealed partial denervation of the left short toe extensor.

He later developed a slight tendency to drop-foot on the left side while walking, but toe movement was almost normal as was the motor power in the short toe extensor. At follow up, 2 years after the accident, conduction velocity in the left peroneal nerve was normal.

*Comment.* In this case the current flow was limited to the upper part of the body as it is unlikely that a strong current at 16,000 V could earth through a leg or a foot without leaving any exter-

nal marks. The lesion of the left peroneal nerve therefore was not electrical in origin. Fractures were excluded by X-rays. Apart from the heaematoma of the left trochanteric region, there were no other external signs of injury to the leg, but despite this, a contusion of the leg is still the most likely explanation for the nerve lesion.

*Case 46.* A 53 year old man, whilst doing repair work on a transformer was traversed by a current at 70,000 V. It arced across to his left arm and earthed through the right foot, causing severe damage to the lower extremity. On admission 14 days later, he had diminished sensation in the sole and toes of the right foot, and complete loss of motor power in the right foot and lower leg. The left leg was uninjured but motor power was somewhat reduced, probably due to sciatica of several years standing.

EMG performed 3 weeks after the accident showed total denervation of the right anterior tibial and gastrocnemious muscles. The short toe extensor was not tested because most of it had sloughed away. When EMG was performed 4 months after the accident, reinnervation was observed in the anterior tibial and gastrocnemious muscles, but motor function of the leg was severely impaired. Further reinnervation was observed one year later, but there was no clinical improvement. Conduction velocities in both arms and left leg were normal during the period of treatment, but the left peroneal nerve was never excitable.

One year after the accident the patient developed a coarse tremor, mainly of the left side with slight rigidity and an expressionless face, suggestive of Parkinsonism. He has now retired, at the age of 55.

*Comment.* The paresis of the right leg can without doubt be explained by thermal injury to the muscles and the peroneal nerve. The EMG findings on the left side can most likely be explained by sciatica unrelated to the electrical trauma.

*Case 47.* A 40 year old man was subjected to a high tension current (6000 V) of very short duration which entered through the right hand when he accidentally touched a live wire with a wrench. The current probably earthed through the right leg where small burns were present. The patient who was rendered unconscious, recovered quickly and felt weakness and numbness of the right arm and hand. On admission to the Burns Unit 30 minutes later, loss of sensation and motor power in that extremity was observed. 2 hours after the accident sensation had partially returned, but the paresis was unchanged. At that time conduction velocities were determined in all four extremities. The values were all within normal limits, but lower in the left ulnar and left peroneal nerves than in the right ones. This was rather unexpected since the current flow was most likely confined to the right side of the patient. 24 hours later sensation was normal and the motor power apparently normal, but somewhat restricted because of pain. The conduction velocities had increased in both median nerves and the difference between right and left side was at that time less pronounced. One week later the conduction velocities were stabilized at relatively normal levels. After excision and skin grafting the patient made an uneventful convalescence, and he is now completely recovered.

*Comment.* The burns did not involve deep structures, and thermal injury to the nerve trunks can probably be excluded. Direct action of the current upon the nerves is therefore possible. There was, however, some discrepancy between the clinical findings and the conduction velocities. As the latter were all within normal limits, this perhaps supports Panse's theory about rapidly reversible changes in the spinal cord, although this would necessitate the current retracing its track after traversing the cord—which seems highly unlikely. There is, of course, the possibility that the current did in fact earth through the left arm without leaving external marks. This would also explain the apparent contradiction of lower conduction velocities in the non-affected (left) arm.

*Case 49.* This patient aged 24, touched the 16,000 V live wire with a pitch-fork, when working in an open freight car at a railway station. The current traversed his body via the hands causing full thickness burns of both wrists and arc burns of the trunk. He was rendered unconscious for a few minutes. Six hours after the accident there were no signs of peripheral nerve injury. Shortly after admission conduction velocities were normal in the main nerves of all four extremities and remained so during treatment, with two exceptions: Four days after the accident, the conduction velocity was reduced to 41 m/sec in the left median nerve and one week later to 40 m/sec in the right median nerve. Two months after the accident conduction velocities were normal in all four extremities.

*Comment.* The normal conduction velocities and the normal neurological findings on the day of admission show that there was no immediate injury of the nerve trunks. The subsequent transient reduction of the velocities was probably due to oedema and eschars preventing a proper placing of the recording electrode.

*Case 71.* This patient, aged 11, was playing on top of an engine at a railway station when the current arced across from the 16,000 V wire to his head and neck and earthed through both feet. He fell unconscious to the ground but recovered after some minutes. On admission 5 hours later, he was found to have deep burns of both soles and extensive arc burns on the head, neck, trunk and

arms. No neurological symptoms were demonstrable until 9 days later when bilateral patellar and ankle clonus developed together with poor coordination in the legs and a positive Babinski sign on the left side. Conduction velocities in both peroneal nerves and EEG were normal 13 days after the accident. After further neurological examination a probable diagnosis of spinal cord lesion was made. The general condition of the patient prevented a more thorough examination and treatment of the burns was considered the first essential. After surgical treatment, the patient was transferred to a hospital near his home for further physiotherapy, and he will be admitted to our unit later for secondary correction. Reports from the local hospital show that he is making progress and that the prognosis regarding the lesion of the cord is considered favourable.

*Comment.* It should be mentioned that neurological signs were not sought daily during the first week, as the findings were normal on admission and on the following day. Limb movements were routinely checked every day as part of the physiotherapy programme and it was not until the 9th day that a neurological lesion was suspected. The aetiology of this lesion of the spinal cord is uncertain. X-rays of the spine were normal, but a partial dislocation with spontaneous reposition cannot be excluded. Medullary concussion is a possibility. Lesions of the spinal cord with similar symptoms after electrical injury have been described and their significance discussed (Koeppen, 1955; Panse, 1955).

### *Group II*

This group has been divided into two sub-groups: (*a*) Patients without and (*b*) Patients with initial abnormal neurological symptoms.

#### *(a) Patients without initial abnormal neurological symptoms*

This group consists of 12 cases. Ten patients had normal neurological findings and normal conduction velocities in the main nerves of all four extremities. EMG examination performed in 3 cases was normal. Two cases however, showed slight abnormalities and will be described in more detail.

*Case 92.* 8 years earlier, when aged 53, this patient sustained moderate arc burns to his face and hands. He was also thrown over by the shock, hurting his left elbow. Later, he noticed a partial loss of sensation in the ring and little fingers of the left hand. This was confirmed on clinical examination. The patient also had a Dupuytren's contracture involving the same fingers. The conduction velocity in the left ulnar nerve was normal but EMG revealed a slightly reduced interference pattern during maximal contraction but no definite loss of motor units.

*Comment.* Current did not pass through the tissues. The burns were superficial and since it is unlikely that the Dupuytren's contracture could be responsible, the obvious cause of the finger anaesthesia is the trauma around the elbow.

*Case 96.* Six years earlier, when aged 20, this patient sustained extensive arc burns, which were relatively deep in the right thenar region. This resulted in considerable scar formation, despite adequate skin grafting. Clinical examination revealed no neurological signs and conduction velocities were normal in both median and ulnar nerves. A slight loss of motor units was however observed in the right opponens pollicis muscle.

*Comment.* Although the main nerve trunks in the arms were spared the immediate thermal injury, it is possible that smaller branches were involved in the burns. The subsequent fibrosis in the nerve branches and their surroundings may explain the EMG findings.

#### *(b) Patients with initial abnormal neurological symptoms*

This group comprises 12 cases. Six patients showed no clinical neurological signs on follow up and conduction velocities were normal in every case. EMG was normal in five cases. Six cases with some abnormalities will be described in more detail.

*Case 39.* Eight years earlier, when aged 37, this patient was injured by direct current at 40,000 V which passed from the left lower arm to the right foot. He was rendered unconscious for several minutes and had convulsions resembling grand mal. A small burn of the left lower arm healed spontaneously. The three medial toes of the right foot had to be amputated and skin cover was obtained by means of an abdominal tube pedicle. On follow-up, clinical examination and EMG were normal, but the conduction velocity was slightly decreased in the left ulnar nerve.

*Comment.* Thermal injury to the nerve cannot be excluded since the current had entered through the volar aspect of the lower arm, leaving a small but deep burn.

*Case 44.* Nine years earlier, when aged 32, this patient was traversed by a current at 10,500 V from the right hand to the left lower leg. On regaining consciousness after 5 minutes he felt paralyzed in the right arm. This

was severely burned and a lower arm amputation had to be performed 20 days later.

The left arm sustained 3rd, degree arc and flame burns and massive scars formed in the antecubital fossa. Excision and skin grafting were later performed at this site on two occasions.

On follow up, movement, power and sensation were all normal in the left arm. Conduction velocity in the median nerve, however, was reduced below the lower limit of normal. EMG showed normal activity in the first dorsal interosseus muscle of the hand and in the left anterior tibial muscle but moderate loss of motor units in the left short toe extensor.

*Comment.* As the left median nerve did not lie in the current path, it is likely that the reduced conduction velocity was due to involvement in the dense scar tissue in the antecubital fossa.

The loss of motor units in the left short toe extensor is more difficult to explain. As the current earthed through the medial aspect of the lower leg, one would have expected the peroneal nerve to have escaped thermal injury, and this is borne out by the normal functioning of the anterior tibial muscle. The possibility of a direct action by the current on branches of the peroneal nerve cannot be excluded.

*Case 52.* Three years earlier when aged 16, the patient had been injured by a current at 16,000 V which entered through both hands and earthed through both feet. He was rendered unconscious for one minute and afterwards had a feeling of numbness throughout his body. He sustained deep burns on both hands, both wrists and the right foot, and a smaller burn on the left foot.

On follow up, there was complete atrophy of the right short toe extensor and pronounced atrophy of the left thenar muscles. EMG showed complete denervation in the former and extensive denervation in the left opponens pollicis muscle. There was moderate loss of motor units in the ulnar nerve innervated muscles of the right hand. Conduction velocity was normal in both ulnar nerves and in the left peroneal nerve, but there was no response in the left median and right peroneal nerves.

*Comment.* The findings in this case can be explained by thermal effects upon muscles and nerves.

*Case 53.* Six years earlier when 28 years old, this patient was exposed to a current at 70,000 V through the same pathway as case 52. Owing to particular conditions mentioned on p. 29, he only sustained minor burns, which were however deep at the contact points. He was rendered unconscious for one minute.

On follow-up, neurological examination was normal as were the conduction velocities in the main nerves of all four extremities. EMG showed slight loss of motor units in both short toe extensors.

*Comment.* Since the burns involved the muscles of the anterior aspect of the lower legs as well as the short toe extensors, the EMG findings can be explained by the thermal injury.

*Case 119.* Five years earlier when aged 24, the patient was traversed by a current at 11,500 V from the right arm to the right leg. He was rendered unconscious for several minutes and on recovery felt his right arm paralyzed. The burns were very extensive and deep and a right upper arm amputation had to be performed.

Clinical examination on follow up revealed slight atrophy and weakness of the right quadriceps muscle. EMG however, showed normal findings in the right leg. Conduction velocities were normal in both peroneal nerves and in the ulnar and median nerves of the left arm.

*Comment.* Although the clinical examination suggested a possible nerve lesion, the neurophysiological findings were normal.

*Case 122.* 13 years earlier when 27 years of age, this patient was traversed by a current at 6000 V from the right scapular region to both feet, whilst working on a pole. He fell down (upon one of his fellows) but did not lose consciousness. Immediately after the shock he felt numbness in both feet and this persisted for several days. Deep burns of the dorsum of the right foot involved the entire short toe extensor as well as parts of the anterior tibial muscle and branches of the peroneal nerve. Three toes had to be amputated.

On follow up, there was marked limitation of movement in the right ankle joint. The short toe extensor could not be palpated. In addition, weakness and atrophy were noticed in the muscles of the left leg, but this resulted from poliomyelitis which the patient had contracted at the age of 19. Conduction velocity was normal in both ulnar and median nerves and in the left peroneal nerve, but there was no response in the right peroneal nerve. EMG showed no activity in the right short toe extensor or in the anterior tibial muscle. On the left side, loss of motor units was observed in the short toe extensor, anterior tibial and soleus muscles.

*Comment.* The findings in the right leg are most likely the result of thermal injury to muscles and nerves. The loss of motor units in the muscles of the left leg cannot be ascribed to the electrical injury but is more likely due to poliomyelitis.

## DISCUSSION AND CONCLUSIONS

This study indicates that permanent damage to peripheral nerves caused by strong electrical currents does not extend beyond the area of local tissue damage. We have no proof that the current selects nerve trunks during its passage through the

tissues, and judged by the clinical appearance of the local lesions encountered in our patients, the current tended to traverse tendons, muscles and blood vessel as readily as nerve trunks. The heat released by the current flow acts upon nerve trunks as well as upon the surrounding tissues and may cause total or partial loss of nerve function. In our patients the most severe nerve lesions, resulting in total loss of function, were directly associated with the thermal effects.

Heat generated in a high tension arc may cause full thickness burns of the skin, but this heat does not, in general, extend into the deeper tissues, thus sparing the nerves.

It has been stated (Panse, 1955; Panse & Menden, 1962) that the peripheral nerves are not more susceptible to electrical injuries than their surrounding tissues, and if the latter recover completely, no degeneration of nerve fibres will occur. This may be right in principle, but the resulting fibrosis and scar formation, both in the nerve trunk and its surroundings, may exert compression upon the nerve fibres and thus give rise to decreased conductibility. This condition was particularly observed in deep burns of the wrist. Deep scar tissue may also form after more superficial burns and can under certain conditions result in impaired nerve function.

Vascular disturbances produced by the current are probably a fairly common cause of loss of nerve function. Clinical experience shows that arterial thrombosis is not an uncommon complication and vasospasm of varying degree may also occur. These processes may so reduce blood supply to the nerve, that partial or total loss of function results. In our patients subjected to neurological and neurophysiological examination we were not able to distinguish alterations of nerve function which could reasonably be attributed to such vascular changes.

Permanent damage to peripheral nerves caused by direct action of the current was not observed with certainty in this material, but it cannot be definitely ruled out.

Transient loss of nerve function may perhaps be thermal in origin, but one is more inclined to the view that it should be considered a direct neuronal action by the current itself. This is particularly suggested in cases where transient loss of nerve function is combined with comparatively superficial lesions. The results obtained in the experimental study in this report support this view. In the animal experiments, deep burns of the posterior extremities were produced by currents strong enough to cause serious injury in man. Typical findings in this study were severe disturbances of function in the sciatic nerve, but these showed a pronounced tendency to reversibility within 30 minutes from the time of shock. This study included a series of experiments designed to evaluate the temperature within the nerve during such current shocks. The temperature rise was never sufficient to produce thermal injury. The neural dysfunction observed was therefore most probably caused by a direct action of the current upon the nerve fibres. The disturbances of nerve function observed in these experiments consisted of a rise in threshold stimulus, a decrease in maximal response amplitude and a drop in conduction velocity. These changes were observed in both motor and sensory fibres, but the former seemed more susceptible than the latter. The experiments were completed by a study in man, on whom comparatively much weaker currents were able to produce similar functional disturbances in the ulnar nerve. These changes were demonstrable in motor fibres only; the strongest current the test subject could stand was insufficient to affect the sensory fibres.

Although the results in animals must be interpreted in relation to their experimental setting, when read in conjunction with the experimental findings in man, they provide fairly conclusive proof that transient loss of function after electrical injury is due to direct action of the current upon the nerve.

Since transient loss of nerve function was observed only to a limited extent in the acute cases, it is likely that the symptoms already had disappeared before the first examination could take place. However, temporary loss of nerve function may have other practical consequences; the patient may be unable to move into safety and thus receive further injury if an automatic cut-out alternately closes and breaks the circuit. Pareses may also result in additional trauma by falling, or by the inability to extinguish the flames if his clothing catches fire.

If the clinical course is otherwise uneventful, the rather mild symptoms of loss of nerve function are usually rapidly reversible, although they occasionally persist for several weeks. In more

extensive, deep burns permanent damage occurs to the nerve trunks; however this damage often seems to be caused either by thermal effects at the time of the injury or by secondary scar formation involving the nerve. In the former case the prognosis is poor indeed, although the possibility of resection and suture should be considered in those cases with a circumscribed lesion of the nerve trunk.

## SUMMARY OF CLINICAL SECTION

### 1. *Incidence*

A review of statistics on electrical injuries, from the turn of the century up to the present time, reveals a relative fall in the accident rate. This is particularly noticeable in Scandinavia, which in proportion to the population, has the highest output of electrical energy in the world. The relative decrease in accident rate is mainly attributed to wider experience and to improvement in safety precautions.

### 2. *Physics*

A survey is given of basic physics with special relevance to electrical trauma. Emphasis is laid upon the fact that tissue damage is related to current flow and not voltage, although the severity of the burn is proportional to the latter. Various concepts of tissue resistance and current pathways through the body are described in more detail.

### 3. *Material*

A series of 122 patients is presented, the vast majority of which were males. In early childhood the distribution was the same in both sexes. Low tension burns were more common in children, whereas most adults were injured by high tension, especially in occupational accidents. Cases admitted for primary and secondary treatment were studied separately.

*Primary cases.* Location and type of burns are described and the severity of the local lesions are studied from different points of view. The series of contact burns has been divided into groups with regard to current pathways through the body, and attempts have been made to correlate the severity of the injury to the resistance of different pathways. The immediate general effects of the current shock has been analysed and lesions to internal organs have been investigated. In low tension accidents, locking to the contacts occasionally occured but damage to internal organs of the trunk was never observed. In high tension accidents unconsciousness was frequently encountered, especially in cases where current had passed through the head; injuries to internal organs were rare and insignificant.

An analysis is made of all the electrical burns and their clinical course. A comparison between low and high tension burns revealed that the latter presented the greater therapeutic problems and accounted for a high degree of disability in many cases. It was also found that as the resistance of the current pathway increased, burns at the contact site tended to be more severe. Thus current flow between both hands or between one hand and one foot often resulted in tissue damage which necessitated amputation. Various other complications such as renal failure, infection, vascular damage, cataract and fracture are reported and commented upon.

Arc burns presented no problems differing from those of thermal burns. Some of them were however extensive and ended fatally in 2 cases.

*Secondary cases.* Eighty per cent of the patients had been injured by low tension. Contracted scars of fingers and lips were the commonest sequelae in this group. Attention is called to the risk of growth disturbance in childrens' fingers if skin grafting is unnecessarily delayed. The fact that relatively large burns of the lip can heal spontaneously leaving only a moderate deformity is shown, and it is recommended that extensive repair should be postponed until the scar tissue has softened.

Severe disability resulting from high tension burns was particularly noticed in cases where current had passed through pathways of high resistance.

### 4. *Treatment*

Different views on the local treatment of electrical burns are discussed. A report is given of our principles in this respect; namely to excise as soon as demarcation occurs and to graft at the earliest possible moment. 84% of the patients have been subjected to follow up examination and

the final functional and cosmetic result evaluated. The need for individualization in treatment is pointed out. Because of the complexity of the electrical injury a centralization of these patients in a Burns Centre is recommended. This enables treatment to be standardized and also encourages research into the obscure problems of burn pathology.

5. *Discussion and conclusions*

The pathogenesis of the electrical burn is discussed and an explanation is offered for the different types of lesion and their localization. The classification of burn injuries in relation to the current pathway is discussed. In the author's opinion the positive correlation between the resistance of current pathway and the severity of the local lesion offers valuable clinical information.

Some complications reputed to be typical in electrical injuries are discussed and evaluated on the basis of the findings in the present series.

Finally, the causes of electrical accidents and their preventability are discussed.

6. *Nerve injuries*

The function of peripheral nerves injured by electricity has been subjected to a more detailed clinical and neurophysiological investigation.

Persistant loss of nerve function was confined almost exclusively to high tension burns, the nerve injury being caused by the immediate thermal effects at the contact sites. However, secondary fibrosis and scar formation was also considered to be of importance. The prognosis regarding function was generally poor, but a circumscribed lesion was found in a few cases where resection and nerve suture was possible. This resulted in partial return of protective sensation.

Transient loss of nerve function probably occurs very often and is likely to be caused by direct action of the current as was shown in the experimental study (Part II) of this report. These functional disturbances set up by the current, mainly in the motor fibres, are rapidly reversible, and complete return of function can be expected provided no thermal or mechanical injury to the nerve has taken place. It is suggested that this transient loss of nerve function is of practical importance as the patient may be unable to move into safety.

# II. EXPERIMENTAL STUDY

ELECTRICAL INJURY TO PERIPHERAL NERVES

## A. *Experiments in cats and rabbits*

### INTRODUCTION

Since experimental animals were first electrocuted in the middle of the eighteenth century, the effects of electricity upon living material have been subjected to intensive research. Attention had been mainly focused upon the mechanism of death and the problems of resuscitation, but from the beginning of this century the local lesions have aroused more interest.

It is difficult to produce graded electrical lesions in test animals under standardized conditions, mainly on account of variations in the resistance of tissues and contacts. This is probably why most experiments have been carried out on a qualitative basis.

Among the wealth of experimental data available, more has been said about morphology than about function, especially as regards peripheral nerves. A common feature in many of these reports is the failure to distinguish between thermal and pure electrical effects.

Various neurological symptoms encountered in some of our patients with electrical injuries provided the incentive for the present experimental study. The purpose was to investigate as quantitatively as possible, the upset in peripheral nerve function of animals experimentally injured by electricity.

#### *Previous investigations*

In 1902 Jellinek described the morphological picture of an electrical injury to the rabbit's sciatic nerve. Alternating current of 2 A at 1000 V was passed for a fraction of a second between the animal's mouth and anus. In two cases, pareses of the hind legs were observed and later histological examination revealed degeneration of the sciatic nerve and changes in the spinal cord, resembling those seen in tabes dorsalis.

Bolognesi (1915) burned the hind legs of rabbits with current from the distribution net but did not record the voltage, amperage or resistance. Sections of the peripheral nerves showed swelling and twisting of the axons and disintegration of the myelin. Similar changes were reported by Pietrusky (1926) who passed direct current at 220 V through the exposed femoral nerve of guinea pigs. Neither Bolognesi nor Pietrusky reported on temperature recordings and the pathological changes were probably caused by heat rather than the current. This is especially likely in the experiments of Bolognesi who used prolonged shocks varying from 30 to 120 seconds. The passage of a strong current through an exposed nerve as performed by Pietrusky, will most likely cause coagulation.

MacMahon (1929) applied repeated, sublethal current shocks to guinea pigs and allowed the animals to survive long enough to produce histological changes. The current was passed from the mouth to one hind leg for five seconds. Temperature recordings were not performed, but since the power was low, the formation of heat in the tissues was probably insignificant. After the third or fourth daily shock, paralysis and diminished sensation was observed in the posterior extremity. The paralyzed animals were sacrificed at varying intervals and a careful histological examination was made in every case. The lesions proved to be almost entirely confined to the nervous system and skeletal muscles. In the peripheral nerves, the individual fibres were swollen and vacuolated with distension of the neurilemma, while the epineurium was infiltrated with leucocytes.

Oda (1936) used a similar technique and passed direct and alternating currents at 40–85 V through the legs of pigeons. On histological examination he found degeneration and resorption of the nerve fibres. It is possible that these changes were caused by direct action of the current itself, but in many cases the power was sufficient to produce deep burns of the legs. The temperature was not recorded.

In 1930 Morrison, Weeks & Cobb published their paper on electrical injuries to mammalian brains. Though not dealing with peripheral nerves this work is mentioned because it is extremely reliable and the experiments were performed under good standardized conditions. Current shocks of varying types and intensities were passed through the brains of guinea pigs, cats and rabbits. The shocks were too small to produce immediate death but strong enough to render the animals unconscious. The brains were later removed for histological examination. The authors concluded that the current produced lesions in the brain and that there was a correlation between the type of lesion and the electrical wave form. An important point in this study is that the complicating factor of heat was eliminated in a convincing manner.

Zolotova & Rivkin (Abstract from Russian by Herman, 1937) exposed dogs to faradic and galvanic currents and examined histologically the peripheral nerves injured by the current. Both types of current produced degeneration in the nerves, whether single or repeated shocks were given. This very brief abstract does not permit a detailed evaluation.

In 1941 Alexander reported to the New York Neurological Society on his experiments on cats. He stated that the critical level which produced permanent damage to nerve tissue was 30 mA per 3 mm of nerve diameter for shocks of 5 seconds duration. Such shocks produced demyelination along their pathway. Further details of these experiments are not available, but Alexander stated that the factor of heat could be ruled out. He expressed the view that the neuronal damage was due to either the current itself or to secondary circulatory disturbances.

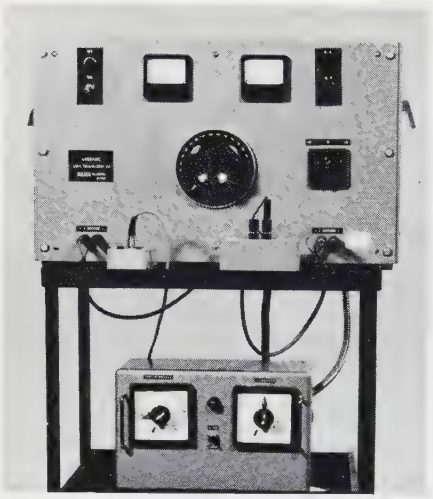
The morphological changes reported in some of the works mentioned above do not permit any definite conclusions. It should be stressed that interpretation of the histological picture of electrical injuries is considered very difficult, and Zeman (1955) pointed out that so-called typical lesions are frequently due to other causes or may even be an artifact. For this reason studies of function, probably give a more realistic approach to the problems. Strangely enough, very few animal experiments seem to have been performed in this field and only three papers will be mentioned.

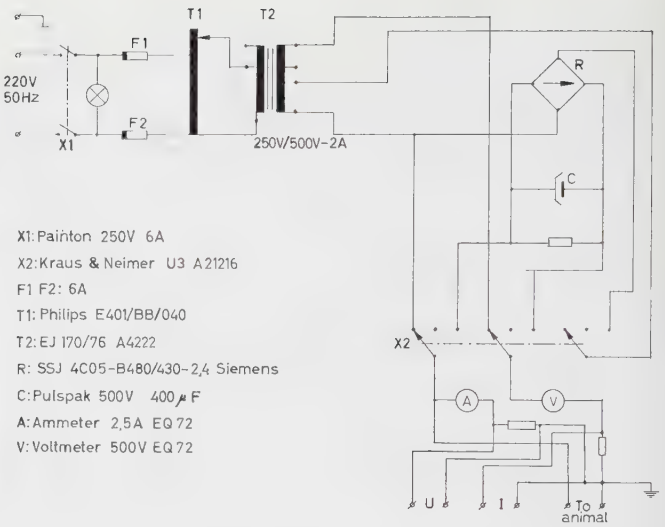
Nothagel (1880) was probably the first to study function in electrically injured nerves. The experiments were carried out under primitive conditions with a rather crude technique. Yet his work is of more than historical interest today. With discharges from Leyden jars he was able to produce reversible paralyses in the femoral and sciatic nerves in rabbits. He also observed that sensory nerve fibres recovered more quickly than motor fibres. Since recovery was complete in all cases he concluded that the disturbed function was not associated with gross anatomical damage.

Urquhart & Noble (1929) passed alternating currents through the exposed sciatic nerve in rabbits and produced a conduction block. The function of the nerve was tested by recording the muscular contractions in the lower leg when the nerve was stimulated. Following the passage of a relatively strong current (20 mA at 150 V) through the nerve a degree of block was established from which recovery did not occur within a reasonable period of time. Temporary blocks were achieved by currents of 5–10 mA at 30–60 V. The authors stated that the blocks were usually reversible, provided no charring of the nerve had taken place.

A paper by Nicolai (1934) should also be mentioned since his technique was partly adopted in the present investigation. Nicolai used nerve-muscle preparations from frogs and passed alternating current through Ringer solution in which the sciatic nerve was immersed. The duration of the current flow varied between 10 and 500 seconds and the amperage was very low. With this technique the author produced persistent functional disturbances in the nerve without causing any thermal injury. Nicolai concluded that the disturbances in function were caused by "specific electrobiological processes".

Useful information has been obtained from the studies mentioned, but the methods have definite limits which prevent any further solution of the problems. Although the cathode ray tube and the oscilloscope were introduced in the early twen-






A B

*Fig. 21.* (A) Power unit and time relay for production of graded electrical injuries. (B) Circuit diagram of power unit.

ties, it is a little surprising that such equipment has not been more widely used for studies in this field.

## PLAN OF INVESTIGATION

The aim of this investigation is to analyse the noxious effects of strong electrical shocks on nerve function in test animals. The study deals with the following main questions:

1. How does a strong current affect factors such as threshold stimulus, amplitude of evoked potentials and conduction velocity?
2. To what extent are the disturbances in nerve function reversible?
3. What rôle does heat play in causing the disturbances?
4. Do certain groups of fibres react differently to electrical injury?
5. What influence has the type and pathway of the current?

## METHODS

Rabbits and cats were chosen for test animals, but as rabbits proved to be vulnerable to the preparation preceding the experiment, cats were preferred.

The study was carried out as "acute experiments" since the preparation of the animal was mutilating and not compatible with continued life.

The electrical injury was produced by a power unit, designed by ASEA (Fig. 21 *a* and *b*). This unit delivers strong direct and alternating currents up to 500 V. The sciatic nerve of the animal was exposed to current shocks of varying intensity, type and duration. Before and after each current shock, nerve function was assessed by recording in the dorsal and ventral nerve roots the response to stimuli applied to the sciatic nerve.

The test stimuli applied to the sciatic nerve were rectangular pulses which were released at a regular frequency of 1/second by synchronisation with the sweep. Pulses with a duration 0.01 –0.1 msec were used, care being taken to keep the duration of the stimulus constant during each experiment. The strength of the pulses could be varied up to 20 V.

The stimulating electrodes were silver hooks with an interpolar distance of 9 mm.

The responses in the roots were transmitted through silver electrodes similar to the stimulating ones. One of the hooks was placed on the crushed end of the root in order to obtain monophasic recordings. A condenser-coupled amplifier (time constant: 5 msec) and a double beam oscilloscope were used for recording. Photographs were taken on bromide paper. Fig. 22 shows the unit used for stimulation and recordings.

Two techniques have been used in producing the electrical injury. In Series I the nerve was

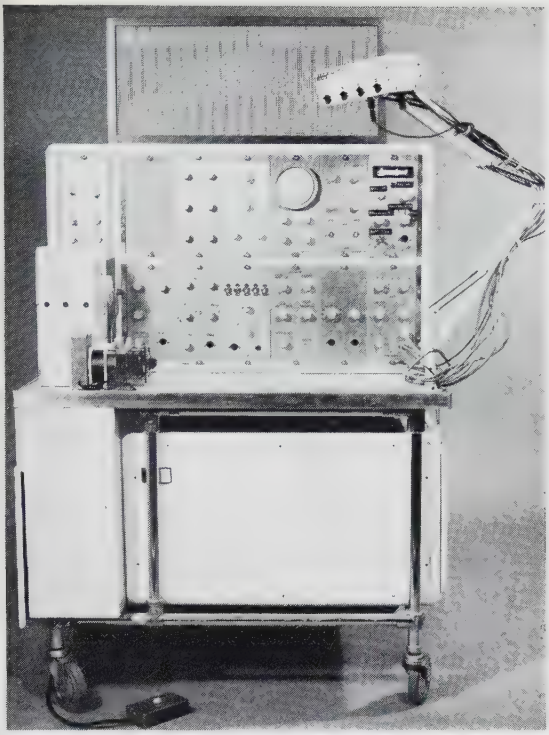


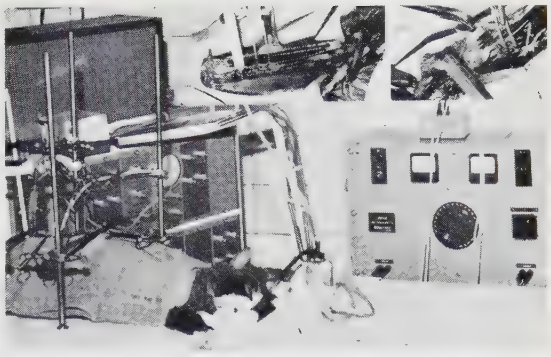
*Fig. 22.* Electronic equipment for stimulation and recording.

injured while immersed in isotonic saline or plasma, simulating the body fluids. In Series II the nerve was injured "in situ".

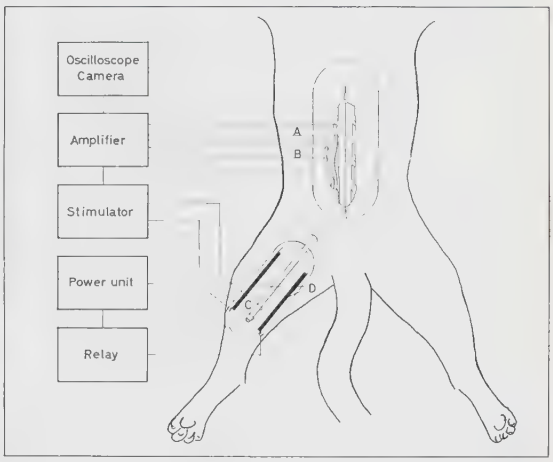
### *Series I*

Operative technique: In this series comprising 15 rabbits and 13 cats the experiments were performed under chloralose-urethane anaesthesia with controlled ventilation through a tracheostomy.

Through a lumbar laminectomy the dorsal and ventral roots of L7 were exposed on one side, severed close to the cord and placed on bipolar recording electrodes in a dorsal pool containing liquid paraffin at 37°C. The sciatic nerve on the same side was exposed, mobilized with caution up to the sciatic foramen and cut at the level of the knee joint. The nerve was immersed in liquid paraffin at 37°C in a small plexiglass vessel and a stimulating electrode was applied to the distal portion of the nerve. Large carbon electrodes were attached to opposite inner walls of the vessel and coupled to the power unit via a time relay. Temperature in the vessel was recorded by a mercury thermometer. The rectal temperature of the animal was checked at regular intervals and maintained at 37°C by means of heating elements. Fig. 23 shows the arrangement of apparatus and test animal.



A




B

*Fig. 23* (A) Arrangement of experimental set-up in Series I. Above left: The sciatic nerve in the plexiglass vessel. Above right: Dorsal paraffin pool with roots and recording electrode. (B) Schematic drawing of experimental set-up. A—dorsal and B—ventral root on recording electrodes. C—stimulating electrode on sciatic nerve. D—carbon electrodes on the walls of the plexiglass vessel. For the sake of clarity connections to earth are omitted.

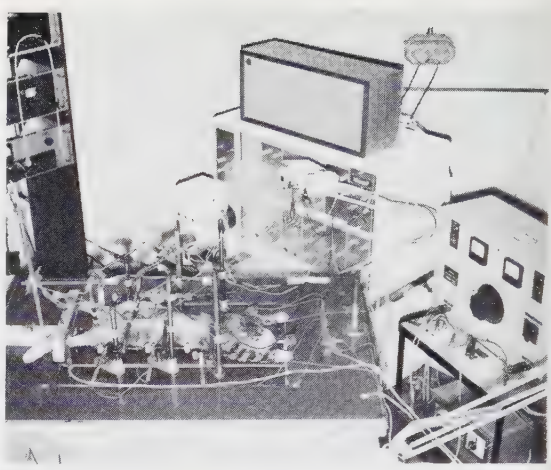
In order to study the properties of the nerve before electrical injury, stimuli of increasing strength were applied to the nerve. The responses evoked in the nerve were recorded from the dorsal and ventral roots. The paraffin in the plexiglass vessel was then replaced by saline or plasma at room temperature and the electrical injury was induced by closing the power circuit for 3 seconds, thus passing the current through the electrolyte and the nerve. The temperature was recorded during the passage of the current. The electrolyte was removed by suction and the vessel

was filled with liquid paraffin at 37°C. The nerve was again stimulated and the responses recorded as before.

The current shocks given to the nerve varied between 140 V and 280 V and each nerve received from 1 to 8 shocks during an experiment. When the current passed through the low resistant electrolyte, the voltage dropped to zero because of the short circuit. Consequently the current intensity became very high, often exceeding 6 A when the fuses melted. In no case was the intensity less than 2.5 A.

*Notes on temperature.* The temperature could not be measured by thermocouples because of artifacts produced by the current shocks. For that reason they were replaced by a highly sensitive mercury thermometer which proved suitable.

In some of the earlier experiments the temperature of the electrolyte rose to 50°C or even more during the current shocks. To eliminate the possible effects of heat, the rest of the experiments were carried out with the nerve immersed in cooled (+4° to +10°C) electrolytes. This prevented the temperature from rising above 40°C. Current flow through the nerve was probably small during the current shocks. The resistance however may have been so high that sufficient heat to damage the nerve could possibly have been generated despite the short exposure of 3 seconds. The fact that the temperature of the electrolyte never exceeded 40°C in the latter part of this series does not rule out a higher temperature within the nerve. As it was impossible to measure this temperature without damaging the nerve, the factor of heat could not be exactly determined.



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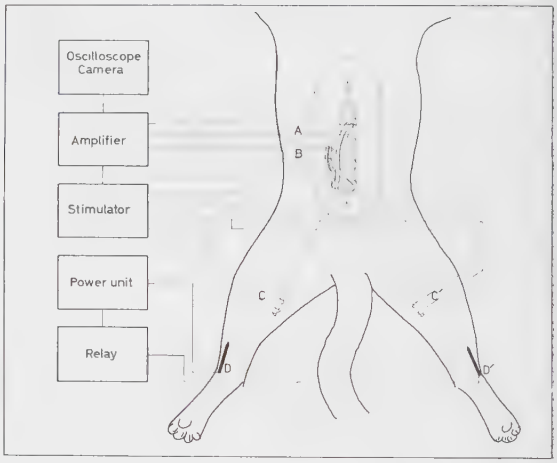


B

*Fig. 24.* (A) Arrangement of experimental set-up in Series II. (B) Schematic drawing of experimental set-up. A—dorsal roots, B—ventral roots. Each pair of roots on one recording electrode. C, C′—stimulating electrodes. D, D′—copper rods conducting current from power unit through animal.

### *Series II*

Operative technique: 32 cats were operated upon, under Nembutal anaesthesia. Free air ways were maintained by tracheostomy.

The dorsal and ventral root of L7 were exposed on both sides and cut close to the cord. The dorsal roots were placed on one bipolar recording electrode (interpolar distance: 5 mm) and the ventral roots on another, thus permitting bilateral recordings. The dorsal paraffin pool was made as before. Both sciatic nerves were exposed, great care being taken to preserve the blood supply. Finally, the nerves were cut at the level of the knee joint. The stimulating electrodes were silver rings 3 mm in diameter with an interpolar distance of 9 mm. The rings were enclosed in a hollow plexiglass cast which insulated the electrodes from the surrounding tissues. One stimulating electrode was applied to the distal portion of each nerve and the skin incisions were closed round the electrodes.

Connections to the power unit were achieved by one copper rod placed subcutaneously in the distal portion of each hind leg, permitting a longitudinal current flow through the nerves. In some cases the copper rods were placed on the ventral and dorsal aspect of the same thigh or in both trochanteric regions in order to study the effects produced by transverse current flow. The copper rods had to be placed subcutaneously to avoid slipping during the current shocks and to eliminate the capacitance of the skin.

To minimize the disturbance caused by violent muscular contractions during the current shocks the animal had

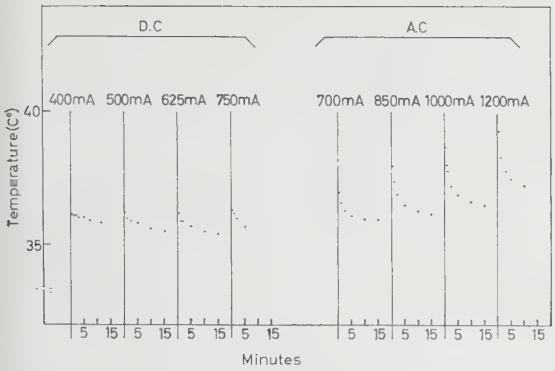


*Fig. 25.* Temperature in the sciatic nerve immediately following. D.C. and A.C.-current shocks of varying intensity. The duration of each shock was 5 seconds.

to be securely fixed to the table with clamps on the spine, pelvis and hind legs. Fig. 24 shows the arrangement of the apparatus and test animal.

With this technique, shocks of 250 to 1700 mA were given at voltages from 200 to 500 V. The duration of the current shock was in most cases 5 seconds, occasionally 10 seconds. Prior to the injury both nerves were stimulated in the same manner as in Series I and the responses were recorded. The electrical injury was induced by closing the power circuit. By simple rearrangement of the electrodes the evoked potentials were easily recorded from the dorsal and ventral roots immediately following the current shocks.

*Notes on temperature.* As in Series I the equipment did not permit simultaneous recordings of the temperature within the nerve. For that reason a separate series of experiments was performed: The animal was secured to the table and the sciatic nerves were exposed in the usual manner. Thermocouples were inserted in the nerves and readings were made on a Cambridge spot galvanometer with an accuracy of $\pm 1$°C. The copper rods were placed subcutaneously in the distal part of the hind legs. Current shocks of the same type, strength and duration were given as in the other experiments and the temperature was checked continuously from the moment the power circuit was opened.

The temperature thus measured in the nerve never exceeded 40°C (Fig. 25).

When A.C. was passed through the animal, oscillographic recordings showed that the voltage was in phase with the current and that the current

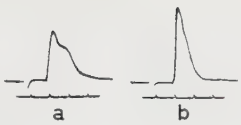


*Fig. 26.* Compound action potentials recorded from the dorsal root (*a*) and ventral root (*b*) of L7 in response to supramaximal stimulation of the cat sciatic nerve. Time in msec.

had the same wave form as when passed through a noninductive or noncapacitive resistance. Under these particular conditions the animal could be considered as a pure ohmic resistance. For that reason the amount of heat released in the tissues during the passage of the current can be computed from Joule's formula:

$$W = R \cdot I^2 \cdot t \cdot 0{,}239 = \text{calories}$$

($R$—resistance of the animal, measured in ohms. $I$—current intensity, measured in ampéres. $t$—duration of contact, measured in seconds. 0.239—Joule's constant.)

The amount of heat released by the current shocks was computed in every case and the highest figure was 1912 calories. If we assume the specific heat of animal tissue to be that of water, 1 cal/g/degree C° and calculate the volume of the animal traversed by the current as approximately 300 cc, the rise in the temperature will be:

$$T = \frac{\text{cal}}{\text{cc}} = \frac{1912}{300} = 6.4^\circ$$

The highest calculated temperature in this series will be: $37^\circ + 6.4^\circ = 43.4^\circ$C.

In fact, the true temperature in the tissues traversed by the current will be somewhat lower owing to the cooling effect of the circulating blood. This is shown experimentally by Binger & Christie (1927).

The measured and the computed temperature seem to be in accordance with each other. It is therefore most unlikely that this moderate rise in temperature would have caused any thermal damage to the nerve.

## RESULTS

### I. *General remarks*

The compound action potential recorded monophasically from the roots in response to maximal

*Fig. 27.* Compound action potentials recorded from the dorsal root of L7 in response to stimuli of increasing strength applied to the cat sciatic nerve. Time in msec.

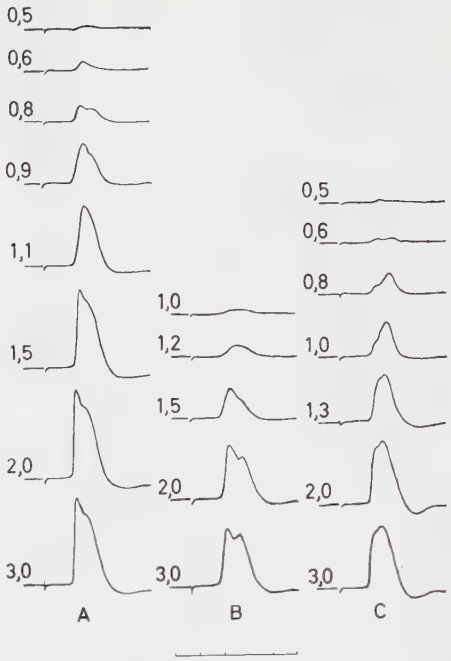


*Fig. 28.* Responses in the dorsal root of L7 to stimuli of increasing strength applied to the cat sciatic nerve. A—before current shock. B—1.5 minutes after A.C. shock at 300 V for 5 seconds. C—3 minutes after A.C. shock at 360 V for 5 seconds. Time in msec.

sciatic stimuli was usually irregular in contour, displaying an initial high elevation with humps of varying magnitude on its descending limb (Fig. 26). The latency of the potential was generally about 1.2 msec. Recordings from the ventral root showed in most cases a shorter latency period than those from the dorsal one. The duration of the potential varied between 1 and 1.5 msec and it was frequently observed that potentials recorded from the ventral root had a shorter duration than those recorded from the dorsal root. The amplitude of the potentials recorded from the dorsal root was usually between 1–2 mV and those from the ventral root 3–5 mV. Occasionally a small later wave was recorded from the ventral root in cats with a latency of about 3 msec.

It is well known that the various components of the compound action potential represent impulse volleys in the A-fibre group, of different conduction velocities and that the amplitude of each elevation reflects the number of fibres activated by the stimulus. Thus the initial deflection represents the fastest alpha fibres whereas the late wave in the ventral root reflects activity in the gamma efferents (Kuffler & *al.* 1951).

In the present study no attempts were made to record activity induced in C-fibres. Fig. 27 shows an example of how the compound action potential in the dorsal root grows with increasing strength of the stimulus applied to the sciatic nerve. An early, small deflection, indicating activity in the fastest fibres appeared at a threshold stimulus strength of 120 mV. This early deflection reached its maximal amplitude at a stimulus strength of about 5 x threshold. At that strength a small deflection had appeared on the declining phase of the potential. No other changes were observed as the stimulus strength was further increased, indicating that all viable afferent A-fibres in the sciatic nerve were excited by the stimulus.

On several occasions it was observed that pressure on the sciatic nerve proximal to the site of stimulation affected the shape of the compound action potential during supramaximal stimulation. This maneouvre temporarily reduced the amplitude of the initial elevation whereas the later part of the potential remained relatively unaffected. This demonstrated the well known fact that the largest and fastest conducting fibres are most easily blocked by pressure (Leksell, 1945). It was soon found that passing noxious electrical currents through the sciatic nerve caused reversible changes in the shape of the compound action potentials, similar to those induced by pressure. Thus the most marked decrease in the evoked potential was usually confined to its initial part (Fig. 28), indicating that the fastest fibres are the most sensitive to electrical injury.

However, no distinction was made between the various components of the evoked potential when

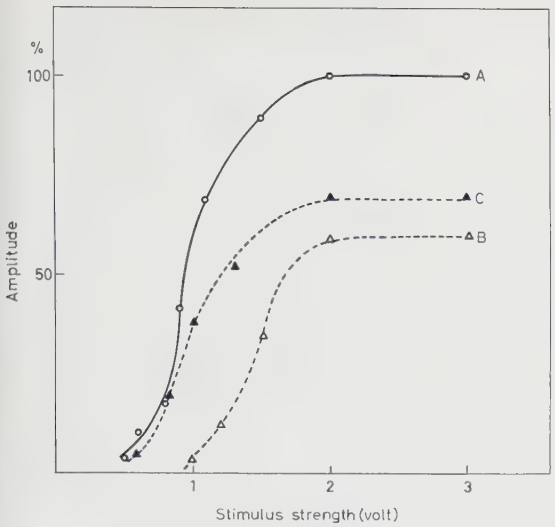


*Fig. 29.* Stimulus strength plotted against the amplitudes of the responses shown in fig. 28. A—before current shock. B—after A.C. shock at 300 V. C—after A.C. shock at 360 V. (It should be noted that a longer period of time was required to get the nerve ready for stimulation after shock C, the nerve thus having more time to recover).

plotting the recovery of response after electrical injury. When making these measurements the peak amplitude served as an index (Fig. 29). Preliminary attempts were made to measure the surface area of the potential but this method was abandoned, partly because it was very tedious, but mainly because the amplitude of the potential proved to be a more sensitive index of impaired nerve function.

II. *Nerve function before and after electrical injury as judged by measuring (a) threshold stimulus, (b) maximal response amplitude and (c) conduction velocity*

(*a*) *Threshold stimulus.* Following a current shock of 150 V in Series I or 500 mA at 250–300 V in Series II a small rise in the threshold was often observed. Stronger shocks (170–180 V in Series I and 750–800 mA at 300–400 V in Series II generally produced a remarkable rise, from about 50 mV initially to 3–4 V after the current shock. In

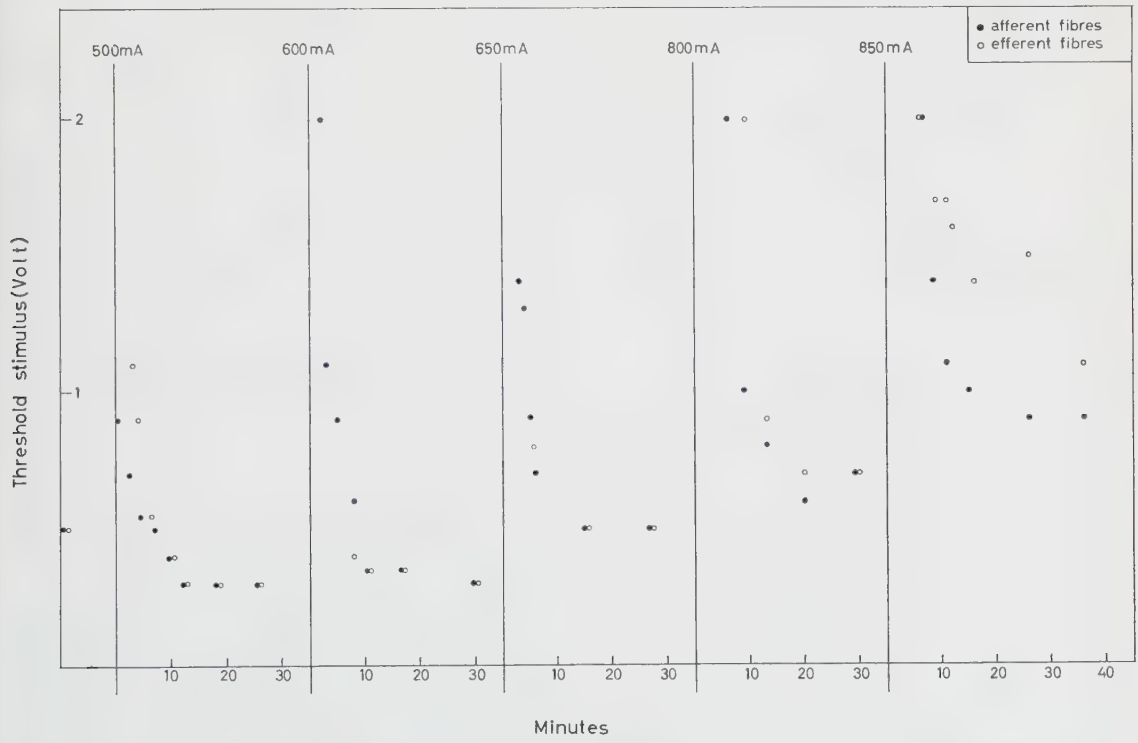


*Fig. 30.* Threshold changes recorded from dorsal and ventral root of L7 before and following 5 successive D.C. shocks of increasing strength, passed through the cat sciatic nerve. The minutes indicate the time after each shock.

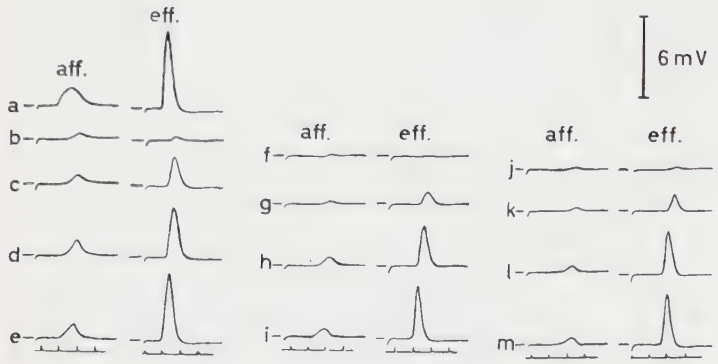


*Fig. 31.* Recordings from dorsal and ventral root of L7 showing changes in the amplitude of responses to supramaximal stimulation of the sciatic nerve, following 3 successive D.C. shocks of increasing strength. a—before current shock, b—5 minutes after 1200 mA for 5 seconds, c—10 minutes after shock, d—15 minutes after shock, e—60 minutes after shock, f—7 minutes after 1500 mA for 5 seconds, g—10 minutes after shock, h—20 minutes after shock, i—70 minutes after shock, j—7 minutes after 1700 mA for 5 seconds, k—10 minutes after shock, l—25 minutes after shock, m—60 minutes after shock. Note the marked reversibility in the efferent fibres. Time in msec.

Series II the threshold could be checked at frequent intervals and a considerable fall was noted a few seconds after the current shock, indicating a pronounced tendency to reversibility. The slope of the threshold followed a hyperbolic curve (Fig. 30), returning to normal in most cases after a few minutes. In some cases however, especially when stronger current shocks had been used, the nerve remained completely inexcitable for a period of 15–20 minutes. The excitability then suddenly returned and the threshold could fall from 3 V to 100 mV over a few seconds. Even stronger current shocks produced a persistent rise in the threshold. The condition was judged persistent if the threshold remained unchanged for more than 30 minutes.

(*b*) *Maximal response amplitude.* Parallel with the rise in threshold there was a decrease in the amplitude of response to supramaximal stimulation. Provided the current shock was not too strong these changes also were reversible, although the amplitude rarely recovered completely (Fig. 31). The amplitude values followed a hyperbolic curve similar to that for threshold values but in the reverse manner (Fig. 32 *a* and *b*). As mentioned above the most marked decrease in the compound action potential was very often confined to its initial part (Fig. 33). The gamma deflection occasionally recorded from the ventral root remained remarkably unaffected by the current shocks.

(*c*) *Conduction velocity.* In most cases an increase of latency was observed after one or more current shocks. The distance between the stimulating electrode and the recording electrodes was measured in every case and the conduction velocity was determined by dividing this distance (in mm.) with the latency (in msec). The quotient gives the conduction velocity in m/second in the fastest fibres. The conduction velocity was frequently greatly reduced by a current shock of sufficient strength to produce changes in the threshold and amplitude. The changes in the conduction velocity were reversible to a similar degree, but the velocity seldom returned to the initial level (Fig. 34). The persistent drop following repeated current shocks was generally small.

Estimations of the latency proved difficult when the induced potential was very small and in these cases the conduction velocity may not be quite correct.

III. *Comparison between sensory and motor fibres*

The changes in nerve function mentioned in the foregoing should be regarded as typical and apply to afferent as well as to efferent fibres.

Some quantitative differences were observed however. In general, the rise in threshold was most pronounced in the efferents and these fibres usually remained inexcitable for a longer period of time than the afferents. On the other hand, the final degree of recovery was similar in both types of fibres.

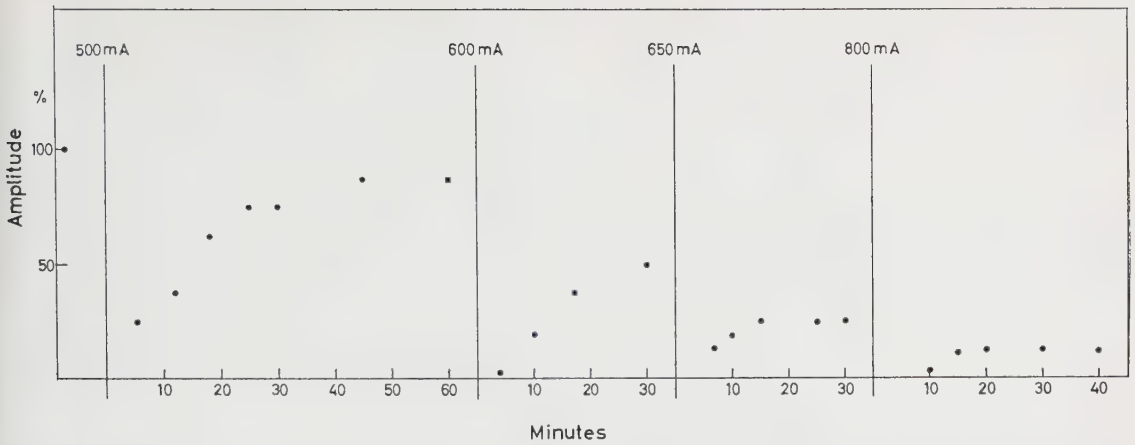


*Fig. 32 A.* Amplitudes of responses to supramaximal stimulation of the cat sciatic nerve, recorded from dorsal root of L7 following 4 successive D.C. shocks of increasing strength. The minutes indicate the time after each shock.

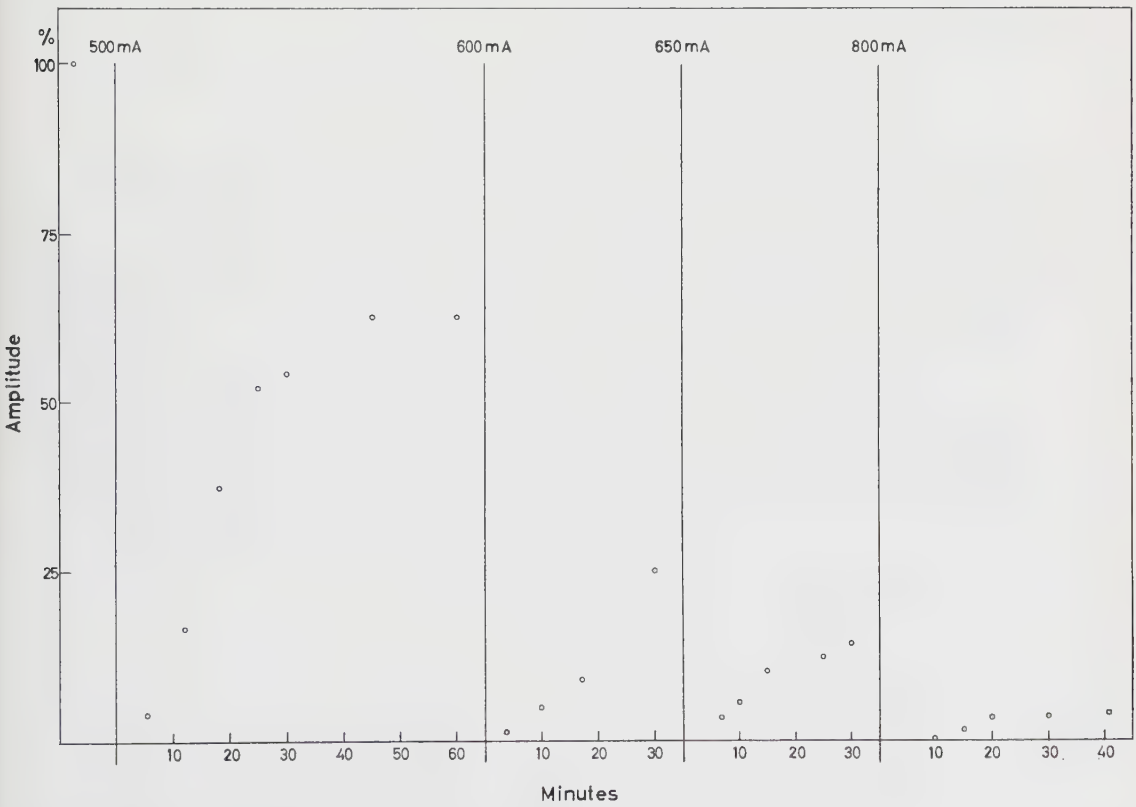


*Fig. 32 B.* Amplitude of responses to supramaximal stimulation of the sciatic nerve, recorded from ventral root of L7 following D.C. shocks of increasing strength.

The amplitude of response to supramaximal stimulation showed a more random behaviour. Immediately after a current shock the efferent fibres showed the greatest decrease in amplitude, but as against this their recovery was shorter. When 30 minutes or more had elapsed from the current shock and the amplitudes had stabilized at constant levels, the efferent fibres showed a more

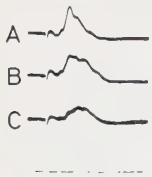


*Fig. 33.* Responses in the dorsal root of L7 to supramaximal stimuli applied to the rabbit sciatic nerve. A—before current shock. B—2 minutes after D.C. shock at 140 V. C—1.5 minutes after D.C. shock at 160 V. Ten superimposed tracings. Time in msec.

persistent decrease. These findings indicate that efferent fibres are possibly more sensitive to electrical shocks than afferent ones. The changes in conduction velocity took place at the same rate in both types of fibres.

In a special series of experiments a comparison was made between skin afferents and muscle afferents. Separate stimuli were applied to the sural and the lateral gastrocnemious nerves and the evoked potentials were recorded from the dorsal roots of L7 and SI. The electrical injury was inflicted in the same way as before. No constant differences could be demonstrated between these two fibre types as regards their susceptibility to electrical injury.

### IV. *Type and pathway of the current*

For practical reasons the majority of the experiments were carried out with D.C. shocks. No qualitative difference between the action of A.C. and D.C. shocks was observed. In Series I it was found that A.C. shocks exerted a somewhat stronger effect upon the nerve function than did D.C. shocks. In Series II (in contradistinction to the results in Series I) the nerve was in every case more sensitive to D.C. shocks, and a constant finding was that much higher power was required when using A.C. shocks if the same effect upon nerve function was to be obtained.

The changes produced in nerve function seemed to be highly dependant upon the pathway of the current. This could be studied in Series II only. Longitudinal current flow through the nerve proved far more effective than transverse current flow. In the latter case considerably more energy was required to produce the same degree of functional disturbance.

## SOURCES OF ERRORS

### *Series I*

These experiments were carried out as a partial "in vitro" study, a method which possesses certain disadvantages and limitations.

The time required to get the nerve ready for stimulation after a current shock was generally 2–4 minutes; the removal of the electrolyte, filling the vessel with paraffin at the proper temperature and replacing the stimulating electrode at the same site constituted a time consuming procedure. This resulted in the insurmountable difficulty of recording nerve function immediately after applying the current shock. Consequently early

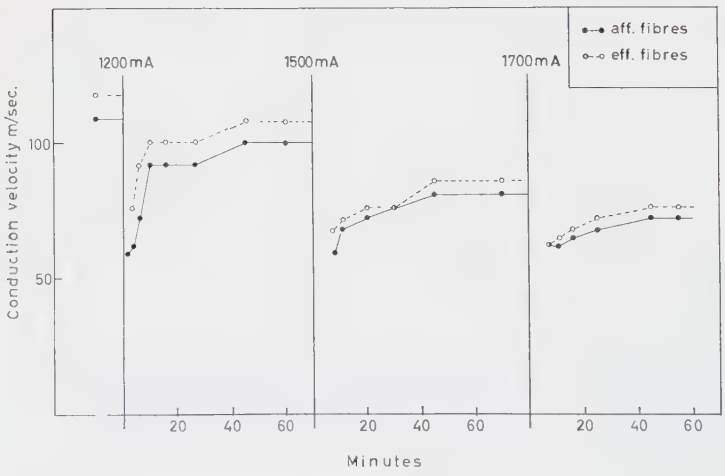


*Fig. 34.* Changes in conduction velocity following D.C. shocks of increasing strength. The curves start a little to the right of each current shock indicated on the diagram, implying a temporary loss of conductivity. See text.

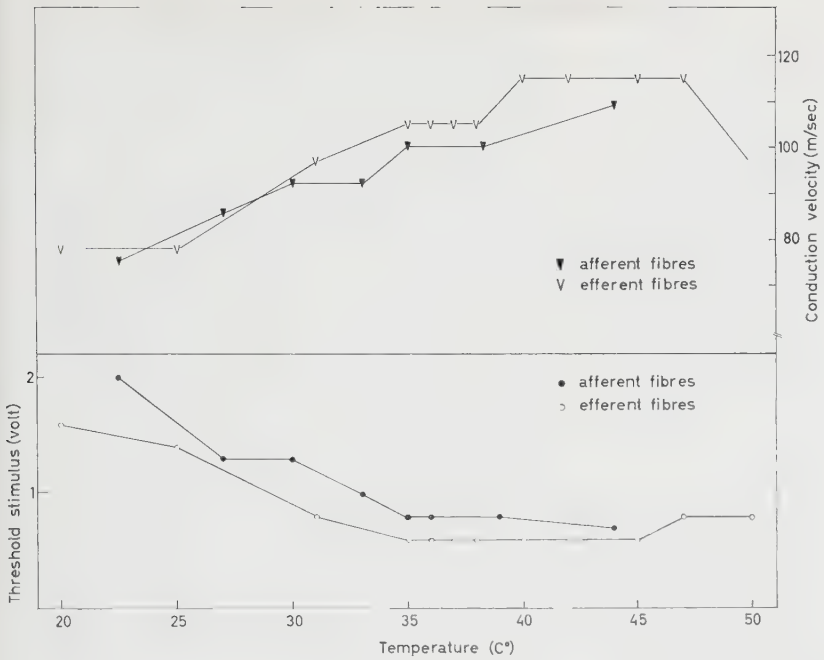


*Fig. 35.* Threshold and conduction velocity in the cat sciatic nerve, tested in liquid paraffin at different temperatures. Note the relatively constant values of threshold and conduction velocity in the range 35°–39°C. Recordings from dorsal and ventral root of L7.

changes in the threshold, latency and amplitude could not be detected.

Harmful effects to nerve function caused by abrupt changes in the temperature of the media surrounding the nerve cannot be excluded. During stimulation and recording the temperature of the paraffin was always kept as close to 37°C as possible. Small deviations from this temperature seem to have only a minor influence upon nerve function, at least as far as threshold and conduction velocity are concerned. This was observed in a special series of experiments where the function of the nerve was tested at different temperatures (Fig. 35).

### *Series II*

These experiments were performed under conditions more closely resembling those of an electrical accident in man. The main source of error in this series was displacement of the roots on the recording electrodes, provoked by muscular contraction during the current shock. Since the slightest displacement of the roots entailed great changes in the recordings, a meticulous fixation of the animal was imperative. By these means it was usually possible to keep the roots in proper position throughout the whole experiment.

After A.C. shocks profuse bleeding into the dorsal paraffin pool was frequently observed, probably caused by a rise in the blood pressure. The blood had a tendency to stick to the roots when it coagulated. The retraction of this coagulum tended to displace the roots on the recording electrodes. This error could never be completely eliminated, but in some cases it was possible to control the bleeding and keep the roots in position long enough to make the recordings.

The resistance between both hind legs varied from 500 to 1500 ohms (average 843 ohm), and was inversely proportional to the size of the cat. Furthermore, the resistance dropped slightly during each current shock, probably because of carbonisation at the points of contact. These factors probably explain the variations in the results.

### *Both series*

Some difficulties in determining the latency were briefly mentioned under Results. An acceptable error in measuring latency would be in the region of $\pm 0.1$ msec. A second complicating factor in computing conduction velocity is the conduction distance of the nerve. At the end of each experiment the whole nerve was exposed and the length measured exactly between the stimulating cathode

and the cut end of the roots. In spite of most meticulous measurement it is reasonable to assume an error of ±2 mm. For these reasons any attempt at greater accuracy in measuring conduction velocity cannot be regarded as reliable.

Following a series of current shocks over a period of 12–14 hours a gradual increase in threshold was often observed. The stepped recording shown in fig. 10 possibly indicates persistent damage to some of the nerve fibres. An alternative explanation is a gradual deterioration of the preparation. Therefore only the first 2 or 3 recordings are considered representative.

## DISCUSSION

This experimental study mainly concerns reversible changes and the term "persistent" should not be taken literally since the studies were performd on "acute animals".

The rise in temperature measured in the sciatic nerve was insignificant and is in keeping with the calculated values. Since this study was carried out under exactly the same conditions as the experiments in Series II it can be assumed that the changes in nerve function in this series were not caused by heat.

Circulatory disturbances provoked by current shocks may be reflected in nerve function (Alexander, 1941; Zeman, 1955; Heidrich *& al.*, 1965). This argument however, does not apply to the observations in the present study. In Series I the nerve was almost totally deprived of its blood supply, yet the compound action potentials obtained pre- and post-shock were essentially the same as observed in Series II. Besides, there is evidence that peripheral nerves can be deprived of their blood supply for several minutes before any major changes in excitability or conductibility occur. The functional changes observed in the present studies are most likely brought about by direct neuronal damage caused by the current itself.

The basic pathology of an electrical injury still remains obscure, but the alteration in shape of the action potential suggests that it resembles a mechanical injury.

Following electrical injury there was a decrease in conduction velocity. As expected, this was most marked in the fastest fibres. In some cases, however, the prolongation of the compound action potential, indicated that the slower fibres were also involved. It has been suggested (Sanders & Whitteridge, 1946; Simpson, 1962; Bannister & Sears, 1962) that conduction velocity is closely related to the properties of myelin. It seems feasable that derangement of the myelin should be associated with changes in conduction velocity. This is in keeping with the histological findings referred to earlier.

The decrease in amplitude of the responses to supramaximal stimulation may be interpreted in different ways: It could be a consequence of altered conduction velocity in individual nerve fibres, making the impulse volleys less synchronized when they reach the recording electrode. This can hardly be the main explanation since the amplitude often changed in parallel with the surface area of the evoked potential.

A few experiments were performed to test whether current shocks could induce a residual injury discharge in individual nerve fibres, thereby giving rise to occlusion phenomena. The sciatic nerve was severed as far proximally as possible and the current shocks were passed from one hind leg to the other while recording from a coaxial electrode inserted into the gastrocnemious muscle. Residual motor activity was never observed. However, the possibility that an occluding residual injury discharge may be induced in sensory nerve fibres cannot be entirely excluded.

The most probable explanation for the decrease in amplitude is that some nerve fibres are functionally blocked and do not respond to the stimulus. Threshold changes may be due to a block in the coarse nerve fibres or to a real rise in the threshold of all nerve fibres. A combination of both factors seems probable. A selective block in the coarse fibres is confirmed by the fact that the initial deflection in the compound action potential was the most susceptible to alteration in amplitude.

Other factors being equal one would expect A.C. shocks to exert the same effect as D.C. shocks, but a comparison of these two current types gave surprising and rather contradictory results. No satisfactory explanation is offered for this peculiar phenomenon.

The results concerning current pathway seem easier to explain. When a current is passed along a nerve, a certain amount of damage would be expected throughout its entire length. With trans-

verse current flow however the lesion is probably confined to a limited segment, thus making the impairment of function correspondingly less.

## CONCLUSION

1. Electrical current shocks of strengths sufficient to cause serious injury in man, produce changes in the function of peripheral nerves in acute test animals.
2. The changes observed in nerve function are reversible to a great extent.
3. These changes are most likely caused by direct neuronal damage induced by the current itself. Heat plays a minor rôle, if any.
4. The degree of dysfunction is dependant upon the type of current and its pathway through the nerve.

# B. *Experiments in man*

S. Blom and O. M. Ugland

Initial symptoms suggesting peripheral nerve damage were sometimes reported by the patients admitted to the Burns Unit after an electrical accident. The objective clinical and neurophysiological findings in emergency cases however, were rather meagre, implying that the functional disturbances had already subsided.

As the animal experiments showed a marked tendency to reversibility of the functional disturbances induced by the current shocks, it seems reasonable that similar rapid changes may also take place in man immediately after an electrical accident. These observations prompted the present study, the purpose of which was to record the initial functional changes which occur in a human peripheral nerve traversed by an alternating current, and also to investigate whether a difference in susceptibility existed between sensory and motor fibres.

## METHODS

The authors and two volunteers served as test subjects. Several experiments were performed on each person.

### *Tetanic stimulation.*

An alternating current of about 10 mA with a frequency of 50 c/s at 15–20 V delivered from a variable transformer was passed through the right lower arm, permitting the current to flow through the ulnar nerve. The active electrode was a padded jack plug soaked in saline which the test subject held in the ulnar nerve sulcus during tetanization. The passive elctrode was a padded lead plate about 3 cm in diameter, soaked in saline and fixed above the wrist. In some cases the current was passed across the arm between two, large, padded, wet lead plates fixed on the ventral and dorsal aspect of the lower arm respectively, care being taken that the nerve lay in the current pathway. Tetanization was obtained by increasing the current intensity up to a strength which the subject could barely stand and maintaining it for 1–1.5 minutes. The current intensity was too weak to cause any burns but strong enough to throw the hand into forceful tetanic flexion contraction. Before and immediately after tetanization the nerve was tested by stimulation; the latency, maximal response amplitude and the conduction velocity being used as an index of the effect.

### *Test Methods*

(*a*) *Motor fibres.* Stimulation: At the moment the tetanizing current was cut off, a supramaximal stimulus was applied alternately to the post-epicondyle jack plug and a similar one held by the investigator over the ulnar nerve at the wrist. The test stimuli were rectangular pulses with a duration of 0.5 msec, given at a frequency of about 1/sec. The distance between the proximal and distal stimulating electrodes was measured. A ground electrode was placed on the wrist, between the stimulating and the recording electrodes.

Recording: A bipolar Disa surface electrode with an interelectrode distance of 23 mm was used, one placed over the tendon and the other over the belly of m. abductor dig. V. The muscle response was amplified and displayed on an oscilloscope. Photographs were made on running bromide paper with a speed of 2.5 cm/sec.

(*b*) *Sensory fibres.* Stimulation: Stimulating electrodes were pipe cleaners soaked in saline and applied round the little finger with an interelectrode distance of about 2 cm. Test stimuli subliminal to motor fibres were given at a frequency of 4–5/sec.

Recordings were made by the same electrode as before, placed in the ulnar nerve sulcus at the elbow. The distance between the stimulating and the recording electrodes was measured. The responses in the sensory fibres were recorded on a CAT-computer, displayed also on another oscillo-

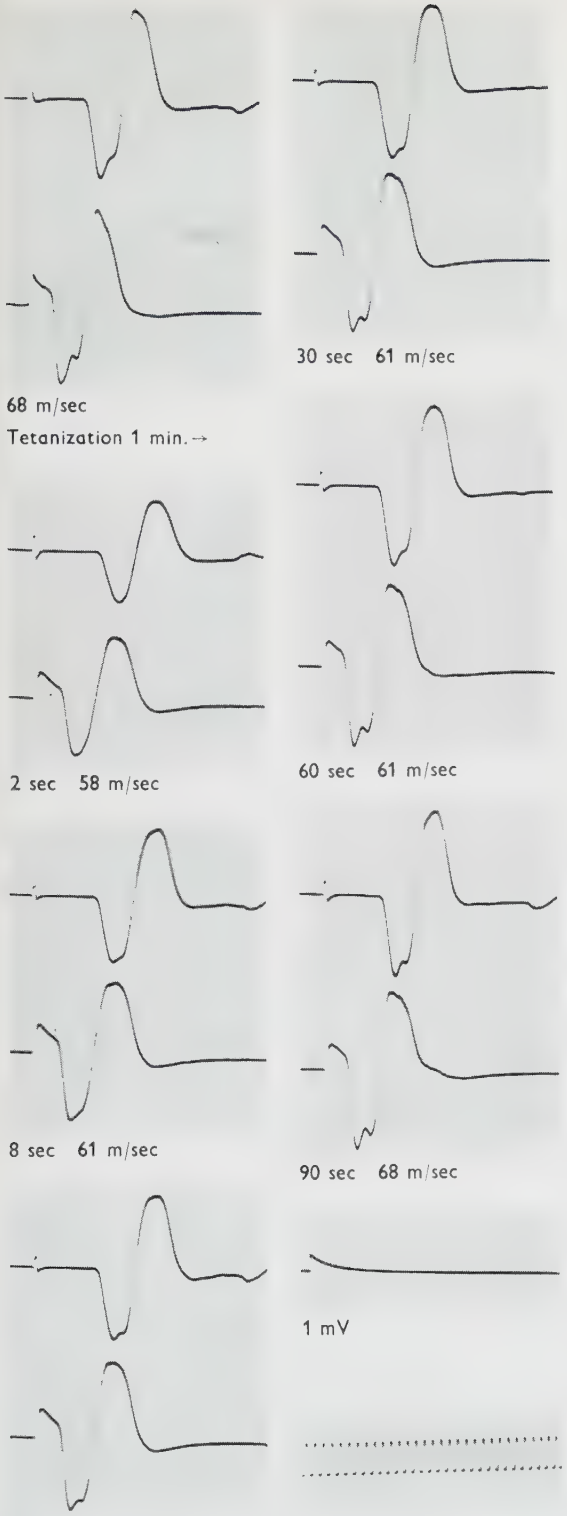


*Fig. 36.*

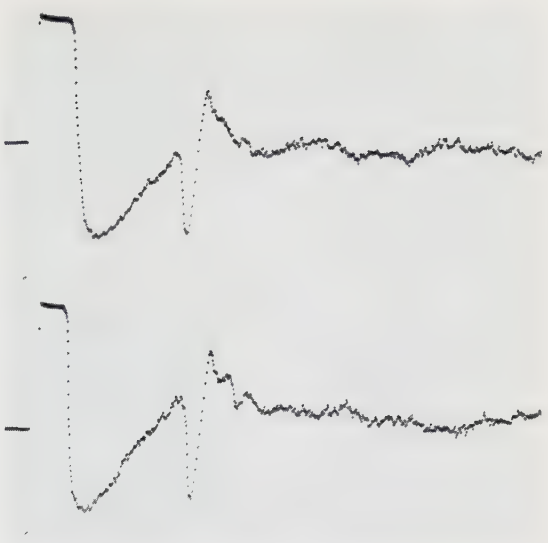

*Fig. 37.* Responses recorded with surface electrodes from the ulnar nerve at the elbow, evoked by ulnar nerve stimuli (subliminal for motor fibres) at the little finger. Stimulus frequency 4–5/sec. Upper tracing before tetanization. Lower tracing immediately after tetanization with 10 mA at 15 V for 70 seconds. Both recordings represent the sum of about 90 responses during 20 sec. The latency is in both recordings 5.75 msec. Distance between stimulating and recording electrode 37 cm.

scope from which photographs were made at a film speed of 3.5 mm/sec.

Switching off the tetanizing current automatically switched on the stimulating and recording unit thus registering the earliest changes in the nerve function.

## RESULTS

The motor fibres showed marked changes of both latency and amplitude (Fig. 36) and a distinct drop in the conduction velocity was also observed. The changes were however so rapidly reversible that the control values were generally reached after about 1 minute. Identical changes could be

*Fig. 36.* Responses recorded with surface electrodes from m. abductor dig. V., evoked by supramaximal stimulation alternately at the elbow and at the wrist. Left upper 2 tracings: before tetanization. In this case a current of 17.5 mA at 18 V was passed across the lower arm for 1 minute. Subsequent recordings made 2, 8, 20, 30, 60 and 90 seconds after tetanization. Note the rapidly reversible changes in amplitude and conduction velocity. Time in all records 1000 cycles/sec.

induced by repeated tetanizations and the recovery time was the same in every case.

The sensory fibres on the other hand, were completely unaffected by the tetanization and no changes were demonstrable in the latency or in the amplitude (Fig. 37). This was a constant finding in every experiment.

## DISCUSSION

In this study the heat factor can be definitely excluded and circulatory disturbances are probably not responsible for the changes observed. A direct neuronal effect by the current may be considered most probable. The current intensity was very low during tetanization, compared with that used in the animal experiments, or indeed with what could be expected in a person exposed to an electrical injury. If we could have increased the intensity of the current through the arm, it is likely that we might have been able to prolong the functional disturbances. This was however not possible without causing injury to the test subjects.

It is remarkable that the functional disturbances were confined to motor fibres only. If tetanization had been stronger we might possibly have been able to observe similar changes in the sensory fibres, as shown in the preceding animal experiments. There is reason to believe that motor fibres in man are more susceptible to electrical injury. This is in keeping with the observations of Blom, Hagbarth & Skoglund (1964), who showed that sensory fibres show less post-tetanic subnormality than motor fibres.

The clinical implications of these studies of the behaviour of animal and human peripheral nerves can be summarized as follows: If loss of function is an initial sign in a peripheral nerve traversed by electrical current but not directly involved in an electrical burn, the loss of function is most likely transient and complete recovery of the function can be expected.

## SUMMARY OF EXPERIMENTAL SECTION

In two series of experiments, the sciatic nerve of cats and rabbits was exposed to graded electrical injury. In Series I the nerve was injured when it was immersed in plasma or physiological saline, simulating the body fluids. In Series II the nerve was injured "in situ". The current shocks used in these experiments were of sufficient strength to cause serious injury in man.

Evoked responses in the sciatic nerve were recorded from the dorsal and ventral roots of L7 before and after each current shock; the threshold stimulus, maximal response amplitude and conduction velocity being used as an index of the effects.

Strong current shocks resulted in a remarkable rise of the threshold, and occasionally the nerve remained inexcitable for a period of 15–20 minutes. The excitability then suddenly returned and the threshold could fall from a considerable height to the initial value over a few seconds. Parallel with the rise in threshold there was a decrease in the amplitude of response to supramaximal stimulation. In general these changes were also reversible, although the amplitude rarely recovered completely. The most marked decrease in the compound action potential was generally confined to its initial part, implying that the fast fibres were more susceptible than the slow ones. These alterations in the compound action potential were similar to those produced by mechanical injury. The conduction velocity was frequently greatly reduced by current shocks sufficient to produce changes in threshold and amplitude. The alterations in conduction velocity were reversible to a similar degree, but the velocity seldom returned to the initial level.

The changes produced in nerve function by electrical shocks were essentially the same in both afferent and efferent fibres, but the results indicated that the latter were more susecptible. On the other hand, no such differences could be demonstrated between skin afferents and muscle afferents.

Some experiments were performed to test whether current shocks could induce a residual injury discharge in individual nerve fibres, thereby giving rise to occlusion phenomena. Residual motor activity was never observed, but the possibility that an occluding residual injury discharge may be induced in sensory nerve fibres cannot be entirely excluded.

The most probable explanation for the decrease in amplitude is that some nerve fibres are functionally blocked and do not respond to the stimulus. Threshold changes may be due to a block in the coarse nerve fibres or to a rise in the

threshold of all nerve fibres. A combination of both factors seems probable. A selective block in the coarse fibres is confirmed by the fact that the initial deflection in the compound action potential was the most susceptible to alterations in amplitude. Alterations in the conduction velocity are probably caused by derangement in the myelin.

The development of the experimental technique included a study to evaluate the temperature in the nerve during the current shocks. The rise in temperature thus measured never exceeded 40°C and is unlikely to be responsible for the changes demonstrated in Series II.

Similar results were obtained whether the nerve was injured whilst immersed in an electrolyte or "in situ". Each method had its advantages and draw-backs, and possible sources of errors are discussed.

Whereas A.C. shocks seemed to have a more pronounced effect in Series I, D.C. shocks proved more effective in Series II. No satisfactory explanation is offered for this peculiar phenomenon. There was however no qualitative difference in the action of these two current types. It was also observed that changes in nerve function were more readily induced by longitudinal current flow than by transverse.

The results obtained in these experiments initiated the subsequent study of a human peripheral nerve exposed to electricity. The authors and two volunteers served as test subjects. An alternating current was passed through the right lower arm, traversing the ulnar nerve and causing forceful tetanic flexion contraction. Before and after tetanization the nerve was tested by stimulation; the latency, maximal response amplitude and the conduction velocity being used as an index of the effect. Motor and sensory fibres were tested separately. Whereas the motor fibres showed marked changes in latency, amplitude and conduction velocity, the sensory fibres were completely unaffected by tetanization. The changes observed in the motor fibres were rapidly reversible and usually returned to normal within 1 minute after tetanization had stopped. Changes in the sensory fibres might possibly have been observed if tetanization had been stronger, but this was not possible without causing injury to the test subjects.

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